Barb
Only
Please

Access DB# 90316

# SEARCH REQUEST FORM

**Scientific and Technical Information Center**

Requester's Full Name: Dawyne C Jones Examiner #: 71299 Date: 28 MAR 03
Art Unit: 1614 Phone Number 30 8-9634 Serial Number: 08/962,040
Mail Box and Bldg/Room Location: 2007, CM1 Results Format Preferred (circle): PAPER DISK E-MAIL
2001 CM1

**If more than one search is submitted, please prioritize searches in order of need.**

**********

Please provide a detailed statement of the search topic, and describe as specifically as possible the subject matter to be searched. Include the elected species or structures, keywords, synonyms, acronyms, and registry numbers, and combine with the concept or utility of the invention. Define any terms that may have a special meaning. Give examples or relevant citations, authors, etc, if known. Please attach a copy of the cover sheet, pertinent claims, and abstract.

Title of Invention: see attached sheet

Inventors (please provide full names): 11

Earliest Priority Filing Date: 15 MAR 1996

*For Sequence Searches Only* Please include all pertinent information (parent, child, divisional, or issued patent numbers) along with the appropriate serial number.*

Please search claim 17, 18 and 22.
and the method of claim 39

Point of Contact:
Barb O'Bryen
Technical Information Specialist
STIC CM1 6A05 308-4291

**********

| STAFF USE ONLY | | Type of Search | | Vendors and cost where applicable | |
|---|---|---|---|---|---|
| Searcher: | BOB | NA Sequence (#) | | STN | str 234 text 255 |
| Searcher Phone #: | | AA Sequence (#) | | Dialog | |
| Searcher Location: | | Structure (#) | 1 | Questel/Orbit | |
| Date Searcher Picked Up: | 4-8-03 | Bibliographic | X | Dr.Link | |
| Date Completed: | 4-8-03 | Litigation | | Lexis/Nexis | |
| Searcher Prep & Review Time: | str 20 / text 20 | Fulltext | | Sequence Systems | |
| Clerical Prep Time: | | Patent Family | | WWW/Internet | |
| Online Time: | 30 / 67 | Other | | Other (specify) | |

PTO-1590 (8-01)

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FILE 'REGISTRY' ENTERED AT 11:05:15 ON 08 APR 2003
USE IS SUBJECT TO THE TERMS OF YOUR STN CUSTOMER AGREEMENT.
PLEASE SEE "HELP USAGETERMS" FOR DETAILS.
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Property values tagged with IC are from the ZIC/VINITI data file
provided by InfoChem.

STRUCTURE FILE UPDATES:    7 APR 2003  HIGHEST RN 502131-66-0
DICTIONARY FILE UPDATES:   7 APR 2003  HIGHEST RN 502131-66-0

TSCA INFORMATION NOW CURRENT THROUGH MAY 20, 2002

  Please note that search-term pricing does apply when
  conducting SmartSELECT searches.

Crossover limits have been increased.  See HELP CROSSOVER for details.

Experimental and calculated property data are now available.  See HELP
PROPERTIES for more information.  See STNote 27, Searching Properties
in the CAS Registry File, for complete details:
http://www.cas.org/ONLINE/STN/STNOTES/stnotes27.pdf

=> d stat que l9

L7                STR
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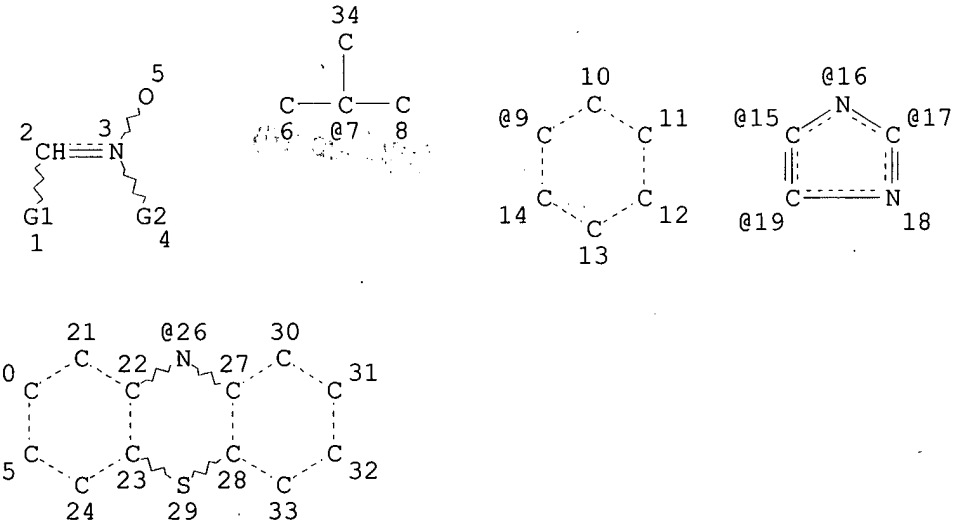


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VAR G1=9/15/16/17/19/26
VAR G2=7/9
NODE ATTRIBUTES:
CONNECT IS E1  RC AT   5
DEFAULT MLEVEL IS ATOM
DEFAULT ECLEVEL IS LIMITED

GRAPH ATTRIBUTES:
RING(S) ARE ISOLATED OR EMBEDDED
NUMBER OF NODES IS  34

STEREO ATTRIBUTES: NONE
L9             1212 SEA FILE=REGISTRY SSS FUL L7

100.0% PROCESSED   6211 ITERATIONS                 1212 ANSWERS
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SEARCH TIME: 00.00.01

=> fil hcapl; d que nos 124; d que nos 126; d que nos 130; d que nos 132; d que nos 133; d que nos 135

FILE 'HCAPLUS' ENTERED AT 11:05:16 ON 08 APR 2003
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FILE COVERS 1907 - 8 Apr 2003 VOL 138 ISS 15
FILE LAST UPDATED: 7 Apr 2003 (20030407/ED)

This file contains CAS Registry Numbers for easy and accurate substance identification.

| | | |
|---|---|---|
| L7 | | STR |
| L9 | 1212 | SEA FILE=REGISTRY SSS FUL L7 |
| L10 | 2014 | SEA FILE=HCAPLUS ABB=ON L9 |
| L11 | 24892 | SEA FILE=HCAPLUS ABB=ON TRAPPING+OLD/CT |
| L12 | 524 | SEA FILE=HCAPLUS ABB=ON L11(L)SPIN |
| L13 | 1001 | SEA FILE=HCAPLUS ABB=ON SPIN TRAPPING+OLD/CT |
| L14 | 563 | SEA FILE=HCAPLUS ABB=ON TOXICITY+NT/CT(L)OXYGEN |
| L15 | 17178 | SEA FILE=HCAPLUS ABB=ON REACTIVE OXYGEN SPECIES/CT |
| L16 | 49447 | SEA FILE=HCAPLUS ABB=ON ANTIOXIDANTS/CT |
| L17 | 19510 | SEA FILE=HCAPLUS ABB=ON OXIDATIVE STRESS, BIOLOGICAL/CT |
| L18 | 170019 | SEA FILE=HCAPLUS ABB=ON OXIDATION/CT |
| L19 | 51371 | SEA FILE=HCAPLUS ABB=ON SKIN, DISEASE+NT/CT |
| L20 | 1 | SEA FILE=REGISTRY ABB=ON 7782-44-7 |
| L21 | 6845 | SEA FILE=HCAPLUS ABB=ON L20(L)ADV/RL |
| L24 | 4 | SEA FILE=HCAPLUS ABB=ON L10 AND ((L12 OR L13 OR L14 OR L15 OR L16 OR L17 OR L18) OR L21) AND L19 |

-oxygen -Role - ADV = adverse effect

| | | |
|---|---|---|
| L7 | | STR |
| L9 | 1212 | SEA FILE=REGISTRY SSS FUL L7 |
| L10 | 2014 | SEA FILE=HCAPLUS ABB=ON L9 |
| L11 | 24892 | SEA FILE=HCAPLUS ABB=ON TRAPPING+OLD/CT |
| L12 | 524 | SEA FILE=HCAPLUS ABB=ON L11(L)SPIN |
| L13 | 1001 | SEA FILE=HCAPLUS ABB=ON SPIN TRAPPING+OLD/CT |
| L14 | 563 | SEA FILE=HCAPLUS ABB=ON TOXICITY+NT/CT(L)OXYGEN |
| L15 | 17178 | SEA FILE=HCAPLUS ABB=ON REACTIVE OXYGEN SPECIES/CT |
| L16 | 49447 | SEA FILE=HCAPLUS ABB=ON ANTIOXIDANTS/CT |
| L17 | 19510 | SEA FILE=HCAPLUS ABB=ON OXIDATIVE STRESS, BIOLOGICAL/CT |
| L18 | 170019 | SEA FILE=HCAPLUS ABB=ON OXIDATION/CT |
| L20 | 1 | SEA FILE=REGISTRY ABB=ON 7782-44-7 |
| L21 | 6845 | SEA FILE=HCAPLUS ABB=ON L20(L)ADV/RL |

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L25       37358 SEA FILE=HCAPLUS ABB=ON  TOPICAL?
L26           2 SEA FILE=HCAPLUS ABB=ON  L10 AND ((L12 OR L13 OR L14 OR L15 OR
                L16 OR L17 OR L18) OR L21) AND L25


L7              STR
L9         1212 SEA FILE=REGISTRY SSS FUL L7
L10        2014 SEA FILE=HCAPLUS ABB=ON  L9
L11       24892 SEA FILE=HCAPLUS ABB=ON  TRAPPING+OLD/CT
L12         524 SEA FILE=HCAPLUS ABB=ON  L11(L)SPIN
L13        1001 SEA FILE=HCAPLUS ABB=ON  SPIN TRAPPING+OLD/CT
L14         563 SEA FILE=HCAPLUS ABB=ON  TOXICITY+NT/CT(L)OXYGEN
L15       17178 SEA FILE=HCAPLUS ABB=ON  REACTIVE OXYGEN SPECIES/CT
L16       49447 SEA FILE=HCAPLUS ABB=ON  ANTIOXIDANTS/CT
L17       19510 SEA FILE=HCAPLUS ABB=ON  OXIDATIVE STRESS, BIOLOGICAL/CT
L18      170019 SEA FILE=HCAPLUS ABB=ON  OXIDATION/CT
L19       51371 SEA FILE=HCAPLUS ABB=ON  SKIN, DISEASE+NT/CT
L20           1 SEA FILE=REGISTRY ABB=ON  7782-44-7
L21        6845 SEA FILE=HCAPLUS ABB=ON  L20(L)ADV/RL
L28         239 SEA FILE=HCAPLUS ABB=ON  L10(L)(THU OR BAC OR PAC OR PKT OR
                DMA)/RL
L29         320 SEA FILE=HCAPLUS ABB=ON  L28 OR (L10 AND PHARMAC?/SC,SX)
L30          11 SEA FILE=HCAPLUS ABB=ON  L29 AND (L12 OR L13) AND ((L14 OR L15
                OR L16 OR L17 OR L18 OR L19) OR L21)

L7              STR
L9         1212 SEA FILE=REGISTRY SSS FUL L7
L10        2014 SEA FILE=HCAPLUS ABB=ON  L9
L11       24892 SEA FILE=HCAPLUS ABB=ON  TRAPPING+OLD/CT
L12         524 SEA FILE=HCAPLUS ABB=ON  L11(L)SPIN
L13        1001 SEA FILE=HCAPLUS ABB=ON  SPIN TRAPPING+OLD/CT
L19       51371 SEA FILE=HCAPLUS ABB=ON  SKIN, DISEASE+NT/CT
L32           3 SEA FILE=HCAPLUS ABB=ON  L10 AND L19 AND (L12 OR L13)


L7              STR
L9         1212 SEA FILE=REGISTRY SSS FUL L7
L10        2014 SEA FILE=HCAPLUS ABB=ON  L9
L14         563 SEA FILE=HCAPLUS ABB=ON  TOXICITY+NT/CT(L)OXYGEN
L15       17178 SEA FILE=HCAPLUS ABB=ON  REACTIVE OXYGEN SPECIES/CT
L16       49447 SEA FILE=HCAPLUS ABB=ON  ANTIOXIDANTS/CT
L17       19510 SEA FILE=HCAPLUS ABB=ON  OXIDATIVE STRESS, BIOLOGICAL/CT
L18      170019 SEA FILE=HCAPLUS ABB=ON  OXIDATION/CT
L19       51371 SEA FILE=HCAPLUS ABB=ON  SKIN, DISEASE+NT/CT
L20           1 SEA FILE=REGISTRY ABB=ON  7782-44-7
L21        6845 SEA FILE=HCAPLUS ABB=ON  L20(L)ADV/RL
L33           2 SEA FILE=HCAPLUS ABB=ON  L10 AND L19 AND ((L14 OR L15 OR L16
                OR L17 OR L18) OR L21)


L7              STR
L9         1212 SEA FILE=REGISTRY SSS FUL L7
L10        2014 SEA FILE=HCAPLUS ABB=ON  L9
L28         239 SEA FILE=HCAPLUS ABB=ON  L10(L)(THU OR BAC OR PAC OR PKT OR
                DMA)/RL
L29         320 SEA FILE=HCAPLUS ABB=ON  L28 OR (L10 AND PHARMAC?/SC,SX)
L34       41883 SEA FILE=HCAPLUS ABB=ON  UV RADIATION+NT,OLD/CT
L35           3 SEA FILE=HCAPLUS ABB=ON  L29 AND L34
```

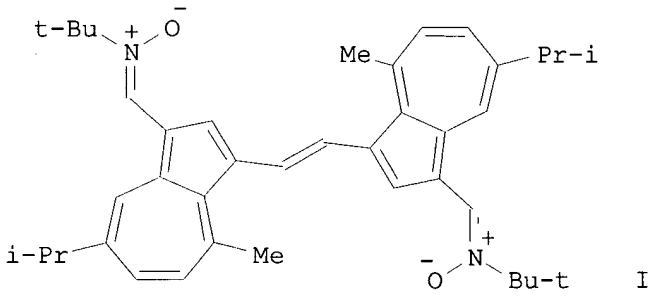
— Roles
THU - Therapeutic use
BAC - Biological activity
PAC - pharmacologic activity
PKT - pharmaco-kinetics
DMA - drug mechanism of action

=> s l24 or l26 or l30 or l32 or l33 or l35

L108 18 L24 OR L26 OR L30 OR L32 OR L33 OR L35

=> d ibib abs hitstr 1-18

L108 ANSWER 1 OF 18 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2002:247206 HCAPLUS
DOCUMENT NUMBER: 136:401379
TITLE: Stilbazulenyl Nitrone (STAZN): A Nitronyl-Substituted Hydrocarbon with the Potency of Classical Phenolic Chain-Breaking Antioxidants
AUTHOR(S): Becker, David A.; Ley, James J.; Echegoyen, Luis; Alvarado, Robert
CORPORATE SOURCE: Departments of Chemistry, Florida International University, Miami, FL, 33199, USA
SOURCE: Journal of the American Chemical Society (2002), 124(17), 4678-4684
CODEN: JACSAT; ISSN: 0002-7863
PUBLISHER: American Chemical Society
DOCUMENT TYPE: Journal
LANGUAGE: English
GI




I

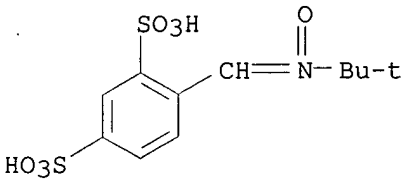
AB Stilbazulenyl nitrone (STAZN), I, a nitronyl-substituted hydrocarbon, is a novel second-generation azulenyl nitrone with significantly enhanced potency as a chain-breaking antioxidant vs. conventional .alpha.-Ph nitrones previously studied as antioxidant therapeutics. A convenient 1H NMR-based assay for assessing the potency of chain-breaking antioxidants showed that STAZN is .apprx.300 times more potent in inhibiting the free radical-mediated aerobic peroxidn. of cumene than is PBN and the exptl. stroke drug NXY-059. Such levels of antioxidant efficacy are unprecedented among archetypal .alpha.-Ph nitrone spin traps. also, STAZN outperforms such classical phenolic antioxidants as BHT and probucol and rivals the antioxidant potency of Vitamin E in a polar medium comprised of 80% cumene and 20% methanol. The Volodarskii electron-transfer mechanism involving the intermediacy of the STAZN radical cation was implicated in attempts to ascertain the basis for the increased potency of STAZN over the three .alpha.-Ph nitrones PBN, S-PBN, and NXY-059.

IT **168021-79-2P**
RL: CPS (Chemical process); PEP (Physical, engineering or chemical process); PRP (Properties); RCT (Reactant); SPN (Synthetic preparation); PREP (Preparation); PROC (Process); RACT (Reactant or reagent)
(antioxidant activity of STAZN vs.; nitronyl-substituted hydrocarbon,

stilbazulenyl nitrone (STAZN), with potency of classical phenolic chain-breaking antioxidants)

RN 168021-79-2 HCAPLUS

CN 1,3-Benzenedisulfonic acid, 4-[[(1,1-dimethylethyl)oxidoimino]methyl]-, disodium salt (9CI) (CA INDEX NAME)



●2 Na

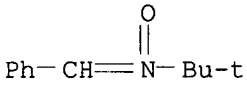
IT **3376-24-7**, PBN **73475-11-3**

RL: RCT (Reactant); RACT (Reactant or reagent)

(antioxidant activity of STAZN vs.; nitronyl-substituted hydrocarbon, stilbazulenyl nitrone (STAZN), with potency of classical phenolic chain-breaking antioxidants)

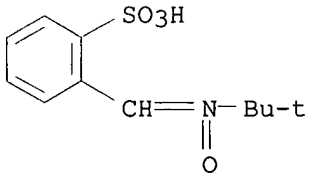
RN 3376-24-7 HCAPLUS

CN 2-Propanamine, 2-methyl-N-(phenylmethylene)-, N-oxide (9CI) (CA INDEX NAME)



RN 73475-11-3 HCAPLUS

CN Benzenesulfonic acid, 2-[[(1,1-dimethylethyl)oxidoimino]methyl]-, sodium salt (9CI) (CA INDEX NAME)



● Na

REFERENCE COUNT: 58 THERE ARE 58 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L108 ANSWER 2 OF 18 HCAPLUS COPYRIGHT 2003 ACS

ACCESSION NUMBER: 2002:168621 HCAPLUS

DOCUMENT NUMBER: 137:134931

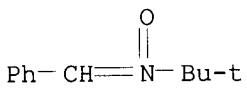
TITLE: The nitrone spin trap PBN alters the cellular response to H2O2: activation of the EGF receptor/ERK pathway

AUTHOR(S): Hassan, Waleed N.; Cantuti-Castelevetri, Ippolita; Denisova, Natalia A.; Yee, Amy S.; Joseph, James A.;

Searched by Barb O'Bryen, STIC 308-4291

Paulson, K. Eric
CORPORATE SOURCE: The Department of Biochemistry, Tufts University School of Medicine, Boston, MA, USA
SOURCE: Free Radical Biology & Medicine (2002), 32(6), 551-561
CODEN: FRBMEH; ISSN: 0891-5849
PUBLISHER: Elsevier Science Inc.
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The nitrone spin trap PBN has been shown to protect neuronal cells from reactive oxygen species both in culture and in vivo. As an approach to understanding the mol. mechanisms by which PBN may function to protect cells, we examd. whether PBN alters the cellular response to reactive oxygen species. H2O2 stimulation of PC-12 cells results in weak activation of both the ERK and JNK signal transduction pathways. PBN pretreatment of PC-12 cells, followed by H2O2 stimulation, results in strong and selective activation of the pro-survival ERK pathway. H2O2 induction of ERK activity in PBN-pretreated cells was shown to be dependent on extracellular Ca+2 influx. Further anal. of the ERK pathway showed that in PBN-pretreated cells, EGF receptor and the adapter protein SHC were phosphorylated in a Ca+2-dependent, ligand-independent manner following H2O2 stimulation. Interestingly, H2O2 stimulation of PBN-pretreated cells results in only 30% of the increase in intracellular Ca+2 as compared to untreated cells following H2O2 stimulation. These data suggest a model in which PBN attenuates H2O2-induced Ca+2 entry, yet magnifies or alters Ca+2 action, resulting in the activation of the EGF receptor/ERK pathway.
IT **7782-44-7D**, Oxygen, reactive species
RL: **ADV (Adverse effect, including toxicity)**; BIOL (Biological study)
(nitrone spin trap PBN alters cellular response to H2O2: activation of the EGF receptor/ERK pathway)
RN 7782-44-7 HCAPLUS
CN Oxygen (8CI, 9CI) (CA INDEX NAME)

IT **3376-24-7**, PBN
RL: **DMA (Drug mechanism of action)**; **PAC (Pharmacological activity)**; **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(nitrone spin trap PBN alters cellular response to H2O2: activation of the EGF receptor/ERK pathway)
RN 3376-24-7 HCAPLUS
CN 2-Propanamine, 2-methyl-N-(phenylmethylene)-, N-oxide (9CI) (CA INDEX NAME)



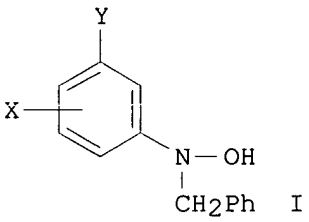
REFERENCE COUNT: 48 THERE ARE 48 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L108 ANSWER 3 OF 18 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2002:40 HCAPLUS
DOCUMENT NUMBER: 136:37389
TITLE: Preparation of N-aryl-N-benzylhydroxyamines as photo-induced DNA-cleaving agents
INVENTOR(S): Hu, Ji-Ru; Tsai, Shu-Jen; Chen, Bu-Luen; Ba, Le-De;

Chen, Wan-Lin
PATENT ASSIGNEE(S): National Science Council, Taiwan
SOURCE: Taiwan, 13 pp.
CODEN: TWXXA5
DOCUMENT TYPE: Patent
LANGUAGE: Chinese
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

| PATENT NO. | KIND | DATE | APPLICATION NO. | DATE |
|---|---|---|---|---|
| TW 378198 | B | 20000101 | TW 1995-84101928 | 19950301 |

PRIORITY APPLN. INFO.: TW 1995-84101928 19950301
OTHER SOURCE(S): CASREACT 136:37389; MARPAT 136:37389
GI

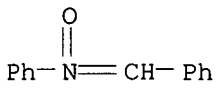


I

AB The present invention discloses a photo-induced DNA-cleaving agent which comprises N-aryl-N-benzylhydroxylamines [I; Y = H, CH3, COOCH3, F, CF3; X = H, 4-CH3, 4-CH3CH2, 2-OCH3, 4-OC6H5, 2-C6H5, 4-F, 2-CH3]. Title compds. I are stable under UV light-free irradn., however I can convert oxygen into hydroxyl radicals under UV irradn; the hydroxyl radicals can then react with DNA to accomplish cleavage of DNA. During the process of cleavage, the UV irradn. initiates and controls the cleavage of DNA. Title cleaving agents, I disclosed in the present invention, have the following advantages, low cost, easy to manipulate, mild reaction condition, high efficiency and suitable for a variety of DNA.

IT **1137-96-8P 19064-77-8P 42790-35-2P**
**115399-97-8P 178923-59-6P 178923-60-9P**
**178923-61-0P 178923-62-1P 178923-63-2P**
**178923-64-3P 178923-65-4P 178923-66-5P**
RL: RCT (Reactant); SPN (Synthetic preparation); PREP (Preparation); RACT (Reactant or reagent)
(prepn. of N-aryl-N-benzyl-hydroxyamines as photo-induced DNA cleaving agents)

RN 1137-96-8 HCAPLUS
CN Benzenamine, N-(phenylmethylene)-, N-oxide (9CI) (CA INDEX NAME)




RN 19064-77-8 HCAPLUS
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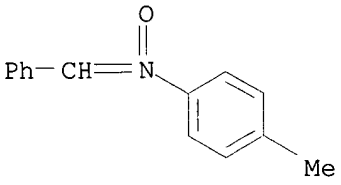
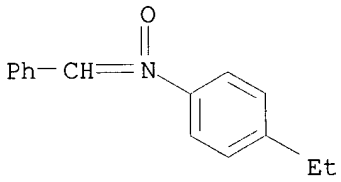
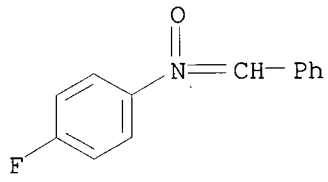


RN 42790-35-2 HCAPLUS
CN Benzenamine, 4-ethyl-N-(phenylmethylene)-, N-oxide (9CI) (CA INDEX NAME)



RN 115399-97-8 HCAPLUS
CN Benzenamine, 4-fluoro-N-(phenylmethylene)-, N-oxide (9CI) (CA INDEX NAME)



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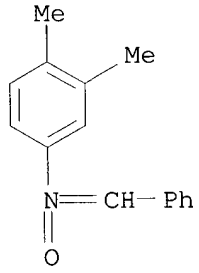

RN 178923-60-9 HCAPLUS
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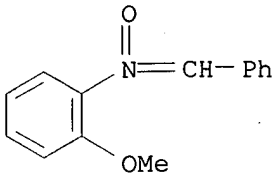
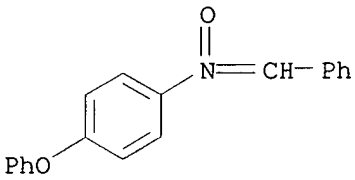
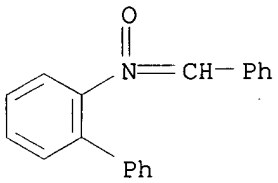


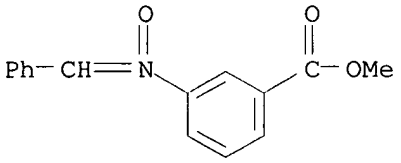
RN 178923-61-0 HCAPLUS
CN Benzenamine, 4-phenoxy-N-(phenylmethylene)-, N-oxide (9CI) (CA INDEX NAME)



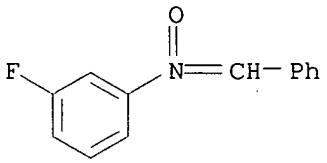
RN 178923-62-1 HCAPLUS
CN [1,1'-Biphenyl]-2-amine, N-(phenylmethylene)-, N-oxide (9CI) (CA INDEX NAME)



RN 178923-63-2 HCAPLUS
CN Benzoic acid, 3-[oxido(phenylmethylene)amino]-, methyl ester (9CI) (CA INDEX NAME)

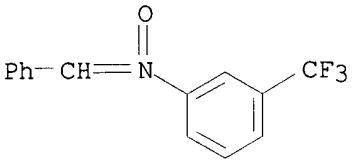


RN 178923-64-3 HCAPLUS
CN Benzenamine, 3-fluoro-N-(phenylmethylene)-, N-oxide (9CI) (CA INDEX NAME)

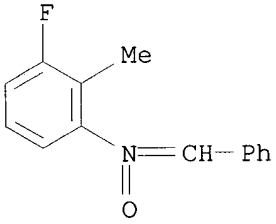


RN 178923-65-4 HCAPLUS
CN Benzenamine, N-(phenylmethylene)-3-(trifluoromethyl)-, N-oxide (9CI) (CA

INDEX NAME)



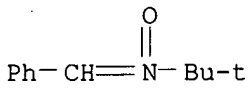
RN 178923-66-5 HCAPLUS
CN Benzenamine, 3-fluoro-2-methyl-N-(phenylmethylene)-, N-oxide (9CI) (CA INDEX NAME)



L108 ANSWER 4 OF 18 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2001:103192 HCAPLUS
DOCUMENT NUMBER: 135:177327
TITLE: UVA-induced oxidative damage in retinal pigment epithelial cells after H2O2 or sparfloxacin exposure
AUTHOR(S): Verna, L. K.; Holman, S. A.; Lee, V. C.; Hoh, J.
CORPORATE SOURCE: Division of Biomedical Sciences, University of California, Riverside, CA, USA
SOURCE: Cell Biology and Toxicology (2000), 16(5), 303-312
CODEN: CBTOE2; ISSN: 0742-2091
PUBLISHER: Kluwer Academic Publishers
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Retinal impairment is one of the leading causes of visual loss in an aging human population. To explore a possible cause for retinal damage in the human population, we have monitored DNA oxidn. in human retinal pigment epithelial (RPE) cells after exposure to hydrogen peroxide (H2O2) or the quinolone antibacterial sparfloxacin. When H2O2- or sparfloxacin-exposed cells were further exposed to UV A (UVA) irradn., oxidative damage to the DNA of these cells was greatly increased over baseline values. This RPE+pharmaceutical-UVA cell system was developed to mimic in vivo retinal degeneration, seen in mouse studies using quinolone and UVA exposure. DNA damage produced by sparfloxacin and UVA in RPE cells could be remedied by the use of antioxidants, indicating a possible in vivo method for prevention or minimization of retinal damage in humans.

IT **3376-24-7**, N-tert-Butyl-.alpha.-phenylnitrone
RL: **BAC (Biological activity or effector, except adverse)**; BSU (Biological study, unclassified); **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(UVA-induced oxidative damage in retinal pigment epithelium after H2O2 or sparfloxacin exposure)
RN 3376-24-7 HCAPLUS
CN 2-Propanamine, 2-methyl-N-(phenylmethylene)-, N-oxide (9CI) (CA INDEX NAME)

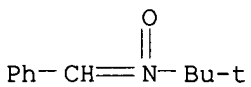


REFERENCE COUNT: 34 THERE ARE 34 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L108 ANSWER 5 OF 18 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2000:895628 HCAPLUS
DOCUMENT NUMBER: 134:189153
TITLE: In vitro and in vivo assessment of the irritation potential of different spin traps in human skin
AUTHOR(S): Fuchs, J.; Groth, N.; Herrling, T.
CORPORATE SOURCE: Zentrum der Dermatologie und Venerologie, Klinikum der J.W. Goethe Universitat, Frankfurt, 60590, Germany
SOURCE: Toxicology (2000), 151(1-3), 55-63
CODEN: TXCYAC; ISSN: 0300-483X
PUBLISHER: Elsevier Science Ireland Ltd.
DOCUMENT TYPE: Journal
LANGUAGE: English

AB No clin. data are available on the acute cutaneous toxicity of spin traps which are frequently used in combination with the ESR technique for detection of free radicals and reactive oxygen/nitrogen species. The purpose of this study was to evaluate the acute dermatotoxicity of the following spin traps in human skin: C-phenyl-N-tert.-Bu nitrone (PBN), C-(4-pyridinyl-N-oxide)-N-tert.-butylnitrone (POBN), 5,5-dimethyl-1-pyrroline-N-oxide(DMPO), 5-diethoxyphosphoryl-5-methyl-1-pyrroline-N-oxide (DEPMPO), diethyldithiocarbamate (DDC) and N-methyl-d-glucamine dithiocarbamate (MGD). The corrosivity of the test substances was first assessed in human skin in vitro by measurement of transcutaneous elec. resistance (TER). In this assay all spin traps were non-corrosive at 500 mM concn. Subsequently cutaneous irritation of the spin traps was detd. at different concns. (50, 250 and 500 mM) in human skin according to a routine four h human patch test in comparison to the standardized irritant sodium laurylsulfate (SLS, 20%). The response was evaluated clin. as well as by a biophys. analyzing transepidermal water loss (TEWL). PBN and DEPMPO caused a transient and weak inflammatory reaction at 500 mM in four of 17 and in two of 17 volunteers, resp. DMPO, POBN, DDC, MGID, and the iron complexes of DDC and MGD were clin. non-irritant at all concns. tested and no delayed-acute inflammatory reactions were obsd. However, the TEWL values were significantly increased by all spin traps except DMPO at 500 mM, indicating disturbed epidermal barrier function. The authors conclude that the spin traps investigated have a low potential to cause acute skin toxicity and may be used safely for in vivo EPR studies in human skin.

IT **3376-24-7**, C-Phenyl-N-tert-butylnitrone
RL: ADV (Adverse effect, including toxicity); BIOL (Biological study)
(In vitro and in vivo assessment of the irritation potential of different spin traps in human skin)
RN 3376-24-7 HCAPLUS
CN 2-Propanamine, 2-methyl-N-(phenylmethylene)-, N-oxide (9CI) (CA INDEX NAME)



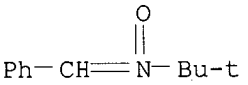
REFERENCE COUNT: 46 THERE ARE 46 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L108 ANSWER 6 OF 18 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2000:364356 HCAPLUS
DOCUMENT NUMBER: 133:172139
TITLE: Do spin traps also act as classical chain-breaking antioxidants? a quantitative kinetic study of phenyl tert-butylnitrone (PBN) in solution and in liposomes
AUTHOR(S): Barclay, L. R. C.; Vinqvist, M. R.
CORPORATE SOURCE: Department of Chemistry, Mount Allison University, Sackville, NB, Can.
SOURCE: Free Radical Biology & Medicine (2000), 28(7), 1079-1090
CODEN: FRBMEH; ISSN: 0891-5849
PUBLISHER: Elsevier Science Inc.
DOCUMENT TYPE: Journal
LANGUAGE: English

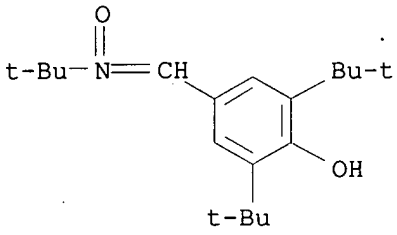
AB Free radical spin traps such as Ph tert-butylnitrone (PBN) are often reported to provide protection of the central nervous system of animal models against free radical damage, and the effects are attributed to its antioxidant activity. The effects of PBN and p-CH3O-PBN were compared with known antioxidants, .alpha.-tocopherol and 2,2,5,7,8-pentamethyl-6-hydroxychroman (PMHC), in quant. kinetic studies of lipid peroxidn. thermally initiated under controlled conditions. Results obtained on the spin traps in org. solvents and in dilinoleoyl phosphatidylcholine (DLPC) bilayers indicated that the spin traps do not act as peroxyl radical trapping antioxidants but rather act only as moderate "retarders" of oxygen uptake at relatively high concn. At low oxygen partial pressures, e.g., 14 torr, which better reflect oxygen partial pressures in biol. systems, PBN provides a more significant redn. in oxygen uptake (up to 50%) by DLPC bilayers but still did not act as a typical antioxidant. However, at low partial pressures, PBN does act cooperatively with PMHC. It is suggested that its role in biol. fluids and tissues may be to extend the suppressed oxidn. by natural antioxidants expected to be present. The combination of antioxidant/spin trap, .alpha.-(3,5-di-tert-butyl-4-hydroxyphenyl)-N-tert-butylnitrone did not exhibit any enhanced antioxidant efficiency compared with the related hindered phenol, 2,6-di-tert-butyl-4-methoxyphenol.

IT **3376-24-7 29211-05-0 40117-28-0**
RL: **BAC (Biological activity or effector, except adverse)**; BSU (Biological study, unclassified); BIOL (Biological study)
(action of spin traps as classical chain-breaking antioxidants and a quant. kinetic study of Ph tert-butylnitrone (PBN) in soln. and in liposomes)

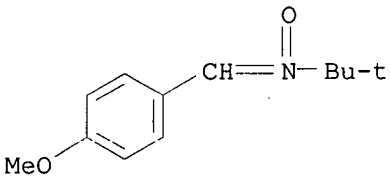
RN 3376-24-7 HCAPLUS
CN 2-Propanamine, 2-methyl-N-(phenylmethylene)-, N-oxide (9CI) (CA INDEX NAME)



RN 29211-05-0 HCAPLUS
CN Phenol, 2,6-bis(1,1-dimethylethyl)-4-[[(1,1-dimethylethyl)oxidoimino]methyl]- (9CI) (CA INDEX NAME)



RN 40117-28-0 HCAPLUS
CN 2-Propanamine, N-[(4-methoxyphenyl)methylene]-2-methyl-, N-oxide (9CI) (CA INDEX NAME)



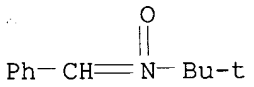
REFERENCE COUNT: 68 THERE ARE 68 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L108 ANSWER 7 OF 18 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2000:8132 HCAPLUS
DOCUMENT NUMBER: 132:218110
TITLE: Spin trapping agent phenyl-N-tert-butylnitrone prevents diisopropylphosphorofluoridate-induced excitotoxicity in skeletal muscle of the rat
AUTHOR(S): Milatovic, D.; Zivin, M.; Hustedt, E.; Dettbarn, W.-D.
CORPORATE SOURCE: School of Medicine, Department of Pharmacology and Neurology, MCS, Vanderbilt University, Nashville, TN, USA
SOURCE: Neuroscience Letters (2000), 278(1,2), 25-28
CODEN: NELED5; ISSN: 0304-3940
PUBLISHER: Elsevier Science Ireland Ltd.
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Indirect evidence suggests that reactive oxygen species (ROS) may mediate muscle fiber necrosis following muscle hyperactivity induced by the anticholinesterase diisopropylphosphorofluoridate (DFP). Pronounced muscle fasciculations and muscle fiber necrosis were seen when acetylcholinesterase (AChE) activity was reduced to <30% of control. The spin trapping agent phenyl-N-tert-butylnitrone (PBN) was used in vivo to directly assess the formation of ROS during DFP (1.75 mg/kg, s.c.)-induced muscle hyperactivity. Pretreatment with PBN (300 mg/kg, i.p.), the concn. necessary for in vivo spin trapping, prevented muscle hyperactivity as well as necrosis and attenuated the DFP-induced AChE inhibition otherwise seen in DFP only treated rats. PBN had no effect when given after fasciculations were established. Muscle exts. from PBN and DFP-treated rats subjected to ESR spectroscopy tested neg. for ROS. While the role of PBN as an antioxidant is well established, its prophylactic effect against excitotoxicity induced by an AChE inhibitor are due to its protection of AChE, an unexpected non-antioxidant action.

IT **3376-24-7**
RL: **BAC (Biological activity or effector, except adverse)**; BSU (Biological study, unclassified); **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(spin trapping agent phenylbutylnitrone prevents

diisopropylphosphorofluoridate-induced excitotoxicity in skeletal muscle)
RN 3376-24-7 HCAPLUS
CN 2-Propanamine, 2-methyl-N-(phenylmethylene)-, N-oxide (9CI) (CA INDEX NAME)



REFERENCE COUNT: 19 THERE ARE 19 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L108 ANSWER 8 OF 18 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 1999:659188 HCAPLUS
DOCUMENT NUMBER: 131:281583
TITLE: Compositions containing a combination of a creatine compound and a neuroprotective compound for the treatment of nervous system diseases
INVENTOR(S): Kaddurah-Daouk, Rima; Beal, M. Flint
PATENT ASSIGNEE(S): Avicena Group, Inc., USA; The General Hospital Corporation
SOURCE: PCT Int. Appl., 81 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

| PATENT NO. | KIND | DATE | APPLICATION NO. | DATE |
|---|---|---|---|---|
| WO 9951097 | A1 | 19991014 | WO 1999-US7340 | 19990402 |
| W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM | | | | |
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| CA 2327095 | AA | 19991014 | CA 1999-2327095 | 19990402 |
| AU 9933803 | A1 | 19991025 | AU 1999-33803 | 19990402 |
| EP 1065931 | A1 | 20010110 | EP 1999-915245 | 19990402 |
| R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI | | | | |
| JP 2002510604 | T2 | 20020409 | JP 2000-541878 | 19990402 |

PRIORITY APPLN. INFO.:
US 1998-80459P P 19980402
US 1999-283267 A 19990401
WO 1999-US7340 W 19990402

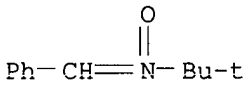
OTHER SOURCE(S): MARPAT 131:281583

AB The invention relates to the use of creatine compd. and neuroprotective combinations including creatine, creatine phosphate, or analogs of creatine, such as cyclocreatine, for treating diseases of the nervous system. Creatine compds. in combination with neuroprotective agents can be used as therapeutically effective compns. against a variety of diseases of the nervous system, e.g. diabetic and toxic neuropathies, peripheral nervous system diseases, Alzheimer disease, Parkinson's disease, stroke, Hungtington's disease, amyotrophic lateral sclerosis, motor neuron disease, traumatic nerve injury, multiple sclerosis, dysmyelination and demyelination disorders, and mitochondrial diseases. The creatine compds.

which can be used in the present method include (1) creatine, creatine phosphate and analogs of these compds. which can act as substrates or substrate analogs for creatine kinase; (2) bisubstrate inhibitors of creatine kinase comprising covalently linked structural analogs of ATP and creatine; (3) creatine analogs which can act as reversible or irreversible inhibitors of creatine kinase; and (4) N-phosphorocreatine analogs bearing nontransferable moieties which mimic the N-phosphoryl group.

IT **3376-24-7**, PBN
RL: **BAC (Biological activity or effector, except adverse)**; BSU (Biological study, unclassified); **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(creatine compd.-neuroprotective compd. combination for treatment of nervous system disease)

RN 3376-24-7 HCAPLUS
CN 2-Propanamine, 2-methyl-N-(phenylmethylene)-, N-oxide (9CI) (CA INDEX NAME)



REFERENCE COUNT: 7 THERE ARE 7 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L108 ANSWER 9 OF 18 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 1999:594909 HCAPLUS
DOCUMENT NUMBER: 131:209129
TITLE: Nitrone-related therapeutics for inhibition of angiogenesis
INVENTOR(S): Narducy, Kenneth W.; Waterbury, Lowell D.; Wilcox, Allan L.
PATENT ASSIGNEE(S): Centaur Pharmaceuticals, USA
SOURCE: PCT Int. Appl., 33 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

| PATENT NO. | KIND | DATE | APPLICATION NO. | DATE |
|---|---|---|---|---|
| WO 9945909 | A2 | 19990916 | WO 1999-US5434 | 19990312 |
| WO 9945909 | A3 | 19991104 | | |
| W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM | | | | |
| RW: GH, GM, KE, LS, MW, SD, SL, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG | | | | |
| CA 2323490 | AA | 19990916 | CA 1999-2323490 | 19990312 |
| AU 9930009 | A1 | 19990927 | AU 1999-30009 | 19990312 |
| EP 1061916 | A2 | 20001227 | EP 1999-911350 | 19990312 |
| R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI | | | | |
| US 6255353 | B1 | 20010703 | US 1999-267510 | 19990312 |
| JP 2002506022 | T2 | 20020226 | JP 2000-535324 | 19990312 |

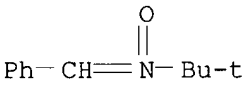
PRIORITY APPLN. INFO.: US 1998-77876P P 19980313
WO 1999-US5434 W 19990312

AB Certain simple chem. agents, referred to herein as nitrone-related

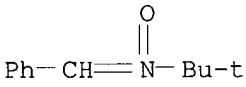
therapeutics (NRTs), when administered to a patient susceptible to neovascularization (angiogenesis), can intervene and inhibit the disease's progress. Methods for therapeutically and prophylactically inhibiting angiogenesis by administering one or more NRTs are disclosed as are pharmaceutical compns. for use in such methods of treating. NRTs useful in these compns. and therapeutic methods are also disclosed.

IT **3376-24-7 3376-24-7D**, analogs **198695-58-8 243457-38-7 243457-40-1 243457-41-2**
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(nitrone-related therapeutics for inhibition of angiogenesis)

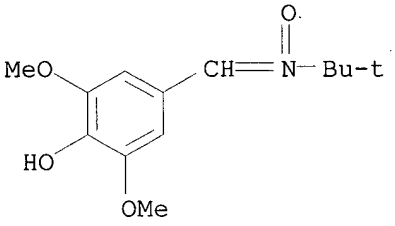
RN 3376-24-7 HCAPLUS
CN 2-Propanamine, 2-methyl-N-(phenylmethylene)-, N-oxide (9CI) (CA INDEX NAME)



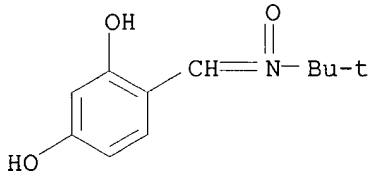
RN 3376-24-7 HCAPLUS
CN 2-Propanamine, 2-methyl-N-(phenylmethylene)-, N-oxide (9CI) (CA INDEX NAME)



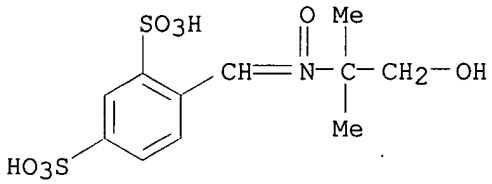
RN 198695-58-8 HCAPLUS
CN Phenol, 4-[[(1,1-dimethylethyl)oxidoimino]methyl]-2,6-dimethoxy- (9CI) (CA INDEX NAME)



RN 243457-38-7 HCAPLUS
CN 1,3-Benzenediol, 4-[[(1,1-dimethylethyl)oxidoimino]methyl]- (9CI) (CA INDEX NAME)

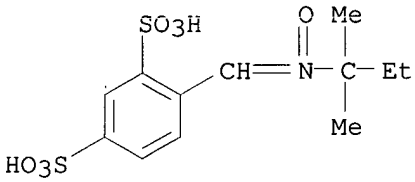


RN 243457-40-1 HCAPLUS
CN 1,3-Benzenedisulfonic acid, 4-[[(2-hydroxy-1,1-dimethylethyl)oxidoimino]methyl]-, disodium salt (9CI) (CA INDEX NAME)



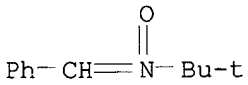
●2 Na

RN 243457-41-2 HCAPLUS
CN 1,3-Benzenedisulfonic acid, 4-[[(1,1-dimethylpropyl)oxidoimino]methyl]-, disodium salt (9CI) (CA INDEX NAME)



●2 Na

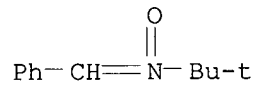
L108 ANSWER 10 OF 18 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 1999:304381 HCAPLUS
DOCUMENT NUMBER: 131:100271
TITLE: Inhibition of endogenous DNA oxidation by spin traps
AUTHOR(S): Zhizhina, G. P.; Mil, E. M.; Binukov, V. I.; Obukhova, L. K.
CORPORATE SOURCE: Emanuel Institute of Biochemical Physics, Russian Academy of Sciences, Moscow, 117977, Russia
SOURCE: Biofizika (1998), 43(1), 35-39
CODEN: BIOFAI; ISSN: 0006-3029
PUBLISHER: Nauka
DOCUMENT TYPE: Journal
LANGUAGE: Russian
AB It was shown that spin trap feeding resulted in a decrease of oxidn. of rat DNA. This is an evidence of free radical mechanism of endogenous DNA oxidn.
IT **3376-24-7**, PBN
RL: BOC (Biological occurrence); BPR (Biological process); BSU (Biological study, unclassified); BUU (Biological use, unclassified); BIOL (Biological study); OCCU (Occurrence); PROC (Process); USES (Uses)
(inhibition of endogenous DNA oxidn. by spin traps)
RN 3376-24-7 HCAPLUS
CN 2-Propanamine, 2-methyl-N-(phenylmethylene)-, N-oxide (9CI) (CA INDEX NAME)



L108 ANSWER 11 OF 18 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 1998:174543 HCAPLUS
DOCUMENT NUMBER: 128:317218
TITLE: Generation of nitric oxide from spin-trapping agents under oxidative conditions
AUTHOR(S): Saito, Kieko; Ariga, Toyohiko; Yoshioka, Hisashi
CORPORATE SOURCE: Graduate School of Nutritional and Environmental Sciences, University of Shizuoka, Shizuoka, 422, Japan
SOURCE: Bioscience, Biotechnology, and Biochemistry (1998), 62(2), 275-279
CODEN: BBBIEJ; ISSN: 0916-8451
PUBLISHER: Japan Society for Bioscience, Biotechnology, and Agrochemistry
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Nitric oxide (NO) generation from the spin-trapping agents, phenyl-tert-butylnitrone (PBN), .alpha.-(4-pyridyl-1-oxide)-N-tert-butylnitrone (POBN) and 5,5-dimethyl-1-pyrroline N-oxide (DMPO), under UV irradn. in the presence of dissolved oxygen and by oxidn. with the Fenton reagent was examd. by using ESR spin-trapping and spectrophotometric methods. A triplet signal at g=2.041 was obsd. after the ferrous complex of dithiocarbamate [Fe(MGD)2] had been added to a soln. of these trapping agents treated with UV irradn. and the Fenton reagent, showing that NO was trapped with Fe(MGD)2. The concn. of nitrite induced from NO was detd. via the Griess reaction to increase with the time of the treatment. It is speculated by ref. to the ESR signal obsd. at the position around g=2.006 that the C=N-double bond might have been cleaved by oxidn., resulting in the formation of a nitroso compd., and that NO was then generated by the fission of the C-N bond of the nitroso compd. NO generated in this way activated guanylate cyclase, from which it can be expected that a spin-trapping agent acts as an NO generator in vivo as well as a free radical scavenger.

IT **3376-24-7**
RL: **BAC (Biological activity or effector, except adverse)**; BSU (Biological study, unclassified); BIOL (Biological study)
(spin-trapping agents as NO generators and radical scavengers)
RN 3376-24-7 HCAPLUS
CN 2-Propanamine, 2-methyl-N-(phenylmethylene)-, N-oxide (9CI) (CA INDEX NAME)



REFERENCE COUNT: 15 THERE ARE 15 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L108 ANSWER 12 OF 18 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 1998:157424 HCAPLUS
DOCUMENT NUMBER: 128:221437
TITLE: **Topical** spin trap composition for the treatment of hair loss and stimulation of hair growth
INVENTOR(S): Proctor, Peter H.
PATENT ASSIGNEE(S): USA

Searched by Barb O'Bryen, STIC 308-4291

SOURCE: U.S., 5 pp., Cont.-in-part of U.S. 5,470,876.
CODEN: USXXAM
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 12
PATENT INFORMATION:

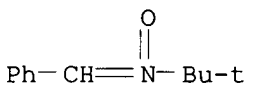
| PATENT NO. | KIND | DATE | APPLICATION NO. | DATE |
|---|---|---|---|---|
| US 5723502 | A | 19980303 | US 1995-465411 | 19950605 |
| US 5352442 | A | 19941004 | US 1993-21970 | 19930224 |
| US 5472687 | A | 19951205 | US 1994-193228 | 19940207 |
| US 5470876 | A | 19951128 | US 1994-229374 | 19940418 |

PRIORITY APPLN. INFO.: US 1985-757131 B2 19850718
US 1986-858050 B2 19860430
US 1987-8186 B2 19870128
US 1988-149720 B2 19880129
US 1993-21970 A2 19930224
US 1994-193228 A2 19940207
US 1994-229374 A2 19940418

AB A compn. and method for ameliorating a cellular dysfunction of a tissue such as the cosmetic treatment of hair loss and stimulation of hair growth are disclosed. The method comprises administering a nitroso or nitrone spin trap such as N-t-butyl-.alpha.-phenylnitrone (PBN) to the affected tissue. A PBN shampoo was prepd. by mixing 0.5 g of PBN in 500 mL of a com. available shampoo. The shampoo was used daily on the scalp for normal shampooing of the hair for a period of from 3 to 6 mo to obtain cosmetic hair growth.

IT **3376-24-7**, N-tert-Butyl-.alpha.-phenylnitrone
RL: BUU (Biological use, unclassified); BIOL (Biological study); USES (Uses)
(**topical** spin trap compn. for treatment of hair loss and stimulation of hair growth)

RN 3376-24-7 HCAPLUS
CN 2-Propanamine, 2-methyl-N-(phenylmethylene)-, N-oxide (9CI) (CA INDEX NAME)



REFERENCE COUNT: 94 THERE ARE 94 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

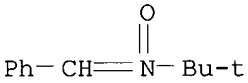
L108 ANSWER 13 OF 18 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 1997:748520 HCAPLUS
DOCUMENT NUMBER: 128:57354
TITLE: MPTP-induced deficits in motor activity: neuroprotective effects of the spin-trapping agent, .alpha.-phenyl-tert-butyl-nitrone (PBN)
AUTHOR(S): Fredriksson, A.; Eriksson, P.; Archer, T.
CORPORATE SOURCE: Department of Psychiatry, University of Uppsala, Uppsala, Swed.
SOURCE: Journal of Neural Transmission (1997), 104(6-7), 579-592
CODEN: JNTRF3; ISSN: 0300-9564
PUBLISHER: Springer
DOCUMENT TYPE: Journal
LANGUAGE: English

AB In Expt. 1, groups of mice were administered either saline or MPTP (2 .times. 30 mg/kg, s.c., sepd. by a 24-h interval) 30 min after being

injected either PBN (15, 50 or 150 mg/kg, s.c., low, medium and high doses, resp.) or L-Deprenyl (0.25 or 10.0 mg/kg, s.c., low and high doses, resp.), the ref. compd. used, or saline. Tests of spontaneous motor activity 14 days later indicated that the MPTP-induced hypokinesia for locomotion and rearing was alleviated by prior administration with PBN (50 or 150 mg/kg) or L-Deprenyl (10.0 mg/kg); lower doses of PBN (15 mg/kg) and L-Deprenyl (0.25 mg/kg) did not affect the MPTP-induced deficits. Dopamine (DA) concns. in the striatum confirmed a more severe loss of DA in the MPTP, PBN(15) + MPTP and Deprenyl(0.25) + MPTP groups than in the control group. Significant protection of DA was obsd. in the PBN(50) + MPTP, PBN(150) + MPTP and Deprenyl(10) + MPTP groups that did not exhibit an hypokinetic behavior. In Expt. 2, the effects of repeated treatment with PBN (50 mg/kg, s.c. over 12 days), post-MPTP, were studied in aged (15-mo-old) and young (3-mo-old) mice. Subchronic administration of PBN increased substantially the motor activity of old and young mice that had received MPTP. Aged control (saline) mice showed an activity deficit compared to young control mice; this deficit was abolished by repeated PBN treatment. The results suggest that moderate-to-high doses of PBN whether injected in a single dose prior to MPTP or subchronically following MPTP injections may afford protective effects against both the functional changes and DA-loss caused by MPTP treatment, possibly through an antioxidant mechanism.

IT **3376-24-7**
RL: **BAC (Biological activity or effector, except adverse)**; BSU (Biological study, unclassified); **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(MPTP-induced deficits in motor activity: neuroprotective effects of the spin-trapping agent, .alpha.-phenyl-tert-butyl-nitrone)

RN 3376-24-7 HCAPLUS
CN 2-Propanamine, 2-methyl-N-(phenylmethylene)-, N-oxide (9CI) (CA INDEX NAME)



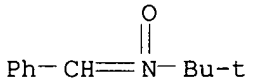
L108 ANSWER 14 OF 18 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 1996:233777 HCAPLUS
DOCUMENT NUMBER: 124:279133
TITLE: Can spin trapping compounds like PBN protect against self-inflicted damage in polymorphonuclear leukocytes?
AUTHOR(S): Seawright, Lorraine; Tanigawa, Mari; Tanigawa, Toru; Kotake, Yashige; Janzen, Edward G.
CORPORATE SOURCE: Natl. Biomed. Cent. Spin Trapping Free Radicals, Oklahoma Med. Res. Foundation, Oklahoma City, OK, 73104, USA
SOURCE: Free Radical Research (1995), 23(1), 73-80
CODEN: FRARER; ISSN: 1071-5762
PUBLISHER: Harwood
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Polymorphonuclear leukocytes (PMNs) have been suggested to be damaged by superoxide radical generated on their own. The protective capacity of a spin trapping compd., phenyl-N-tert-Bu nitrone (PBN) was evaluated for this damage which occurs after the induction of superoxide generation. The life span of PMNs after superoxide generation was measured in the presence of PBN using the cell counting method, and effects of PBN on the amt. of superoxide generated were quantitated using both cytochrome c redn. and spin trapping with DMPO. Results indicated significant extension of life span when PBN was present, and the extension was dose

dependent. However, the magnitude of life span extension was not as large as expected from the decrease of superoxide generation. Possible mechanisms for the protection of PMNs by PBN are discussed.

IT **3376-24-7**, PBN
RL: **BAC (Biological activity or effector, except adverse)**; BSU (Biological study, unclassified); **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(can spin trapping compds. like PBN protect against self-inflicted damage in polymorphonuclear leukocytes)

RN 3376-24-7 HCAPLUS
CN 2-Propanamine, 2-methyl-N-(phenylmethylene)-, N-oxide (9CI) (CA INDEX NAME)

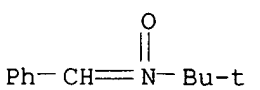


L108 ANSWER 15 OF 18 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 1995:268480 HCAPLUS
DOCUMENT NUMBER: 122:50124
TITLE: Lipid peroxidation by UV or x-ray irradiation and its control
AUTHOR(S): Mio, Takaya; Takaya, Ikuo; Sumino, Kimiaki
CORPORATE SOURCE: school of Medicine, Kobe University, Japan
SOURCE: Nippon Iyo Masu Supekutoru Gakkai Koenshu (1994), 19, 119-24
CODEN: NIMKEN; ISSN: 0916-085X
PUBLISHER: Nippon Iyo Masu Supekutoru Gakkai
DOCUMENT TYPE: Journal
LANGUAGE: Japanese

AB For identification of oxidized lipids, tissues were irradiated with UV-B,C or x-rays and oxidized lipids were analyzed by GC/MS. Cholesta-3,5-diene levels were specifically decreased in the CHCl3-MeOH ext. of red cell membranes in a type II diabetes patient. Cholesta-3,5-diene in CHCl3 soln., irradiated with UV-B,C or x-rays was oxidized to cholestatriene, cholestadiene oxide, cholestene oxide, and cholestane dioxide as lipid peroxidn. products. Added propentofylline and idebenone inhibited lipid peroxidn., whereas N-tert-butyl-.alpha.-Ph nitrone (PBN) combined with Fe2+ accelerated it. The yield of oxidized cholesterol in the presence of PBN was twice that in its absence.

IT **3376-24-7**, N-tert-Butyl .alpha.-phenyl nitrone
RL: **BAC (Biological activity or effector, except adverse)**; BSU (Biological study, unclassified); **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(combination with Fe2+; lipid peroxidn. by UV or x-ray irradn. and its control)

RN 3376-24-7 HCAPLUS
CN 2-Propanamine, 2-methyl-N-(phenylmethylene)-, N-oxide (9CI) (CA INDEX NAME)



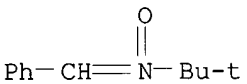
L108 ANSWER 16 OF 18 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 1994:23472 HCAPLUS
DOCUMENT NUMBER: 120:23472

TITLE: Alpha-phenyl-tert-butyl-nitrone (PBN) attenuates hydroxyl radical production during ischemia-reperfusion injury of rat brain: an EPR study
AUTHOR(S): Sen, Souvik; Phillis, John W.
CORPORATE SOURCE: Sch. Med., Wayne State Univ., Detroit, MI, 48201, USA
SOURCE: Free Radical Research Communications (1993), 19(4), 255-65
CODEN: FRRCEX; ISSN: 8755-0199
DOCUMENT TYPE: Journal
LANGUAGE: English

AB .alpha.-Phenyl-tert-butyl-nitrone (PBN) a spin adduct forming agent is believed to have a protective action in ischemia-reperfusion injury of brain by forming adducts of oxygen free radicals including .bul.OH radical. ESR has been used to both detect and monitor the time course of oxygen free radical formation in the in vivo rat cerebral cortex. Cortical cups were placed over both cerebral hemispheres of methoxyflurane anesthetized rats prepd. for four vessel occlusion-evoked cerebral ischemia. Prior to the onset of sample collection, both cups were perfused with artificial cerebrospinal fluid (aCSF) contg. the spin trap agent .alpha.-(4-pyridyl-1-oxide)-N-tert butylnitrone (POBN 100 mM) for 20 min. In addn. 50 mg/kg BW of POBN was administered i.p. 20 min prior to ischemia in order to improve the authors' ability to detect free radical adducts. Cup fluid was subsequently replaced every 15 min during ischemia and every 10 min during reperfusion with fresh POBN contg. CSF and the collected cortical superfusates were analyzed for radical adducts by EPR spectroscopy. After a basal 10 min collection, cerebral ischemia was induced for 15 or 30 min (confirmed by EEG flattening) followed by a 90 min reperfusion. .bul.OH radical adducts (characterized by six line EPR spectra) were detected during ischemia and 90 min reperfusion. No adduct was detected in the basal sample or after 90 min of reperfusion. Similar results were obtained when diethylenetriaminepenta-acetic acid (100 .mu.M; DETAPAC) a chelating agent was included in the artificial CSF. Systemic administration of PBN (100 mg/kg BW) produced a significant attenuation of radical adduct during reperfusion. A combination of systemic and **topical** PBN (100 mM) was required to suppress .bul.OH radical adduct formation during ischemia as well as reperfusion. PBN free radical adducts were detected in EPR spectra of the lipid exts. of PBN treated rat brains subjected to ischemia/reperfusion. Thus this study suggests that PBN's protective action in cerebral ischemia/reperfusion injury is related to its ability to prevent a cascade of free radical generation by forming spin adducts.

IT **3376-24-7**
RL: BIOL (Biological study)
(oxygen radical adduct formation by, neuroprotectant activity during brain ischemia in relation to)

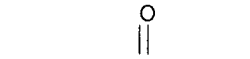
RN 3376-24-7 HCAPLUS
CN 2-Propanamine, 2-methyl-N-(phenylmethylene)-, N-oxide (9CI) (CA INDEX NAME)



L108 ANSWER 17 OF 18 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 1993:462221 HCAPLUS
DOCUMENT NUMBER: 119:62221
TITLE: Protection against oxidative damage to CNS by .alpha.-phenyl-tert-butyl nitrone and other spin-trapping agents: A novel series of nonlipid free radical scavengers

Searched by Barb O'Bryen, STIC 308-4291

AUTHOR(S): Floyd, Robert A.; Carney, John M.
CORPORATE SOURCE: Mol. Toxicol. Res. Program, Oklahoma Med. Res. Found., Oklahoma City, OK, 73104, USA
SOURCE: Emerging Strategies Neuroprot. (1992), 252-72.
Editor(s): Marangos, Paul J.; Lal, Harbans.
Birkhaeuser: Boston, Mass.
CODEN: 59CZA9
DOCUMENT TYPE: Conference; General Review
LANGUAGE: English
AB A review with 18 refs.
IT **3376-24-7**
RL: BIOL (Biological study)
(oxidative damage to central nervous system prevention by)
RN 3376-24-7 HCAPLUS
CN 2-Propanamine, 2-methyl-N-(phenylmethylene)-, N-oxide (9CI) (CA INDEX NAME)



L108 ANSWER 18 OF 18 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 1993:116773 HCAPLUS
DOCUMENT NUMBER: 118:116773
TITLE: spin trapping agents for the treatment of diseases associated with oxidation of lipids and proteins
INVENTOR(S): Carney, John M.; Floyd, Robert A.
PATENT ASSIGNEE(S): Oklahoma Medical Research Foundation, USA; University of Kentucky Research Foundation
SOURCE: PCT Int. Appl., 52 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 4
PATENT INFORMATION:

| PATENT NO. | KIND | DATE | APPLICATION NO. | DATE |
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| WO 9222290 | A1 | 19921223 | WO 1992-US5194 | 19920618 |
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| RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, DE, DK, ES, FR, GA, GB, GN, GR, IT, LU, MC, ML, MR, NL, SE, SN, TD, TG | | | | |
| AU 9222614 | A1 | 19930112 | AU 1992-22614 | 19920618 |
| AU 672364 | B2 | 19961003 | | |
| EP 590072 | A1 | 19940406 | EP 1992-914539 | 19920618 |
| EP 590072 | B1 | 20011205 | | |
| R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, MC, NL, SE | | | | |
| AT 209908 | E | 20011215 | AT 1992-914539 | 19920618 |
| CA 2111836 | AA | 19921223 | CA 1992-2111836 | 19921223 |
| US 5622994 | A | 19970422 | US 1994-212800 | 19940315 |
| US 6002001 | A | 19991214 | US 1997-969344 | 19971128 |
| US 6403627 | B1 | 20020611 | US 1999-357297 | 19990720 |

PRIORITY APPLN. INFO.:
US 1991-716952 A2 19910618
US 1989-422651 A2 19891017
US 1990-589177 B2 19900927
WO 1992-US5194 A 19920618
US 1993-52870 B1 19930426
US 1994-212800 A2 19940315
US 1994-167900 B1 19940729

Searched by Barb O'Bryen, STIC 308-4291

US 1997-969344 A1 19971128

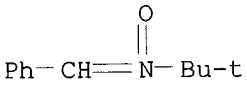
OTHER SOURCE(S): MARPAT 118:116773

AB In the preferred embodiment of the invention, compns. for treating tissue damage from ischemia contain .alpha.-Ph tert-Bu nitrone (I), or active derivs. thereof, in a suitable pharmaceutical carrier. Other preferred spin-trapping agents include 5,5-dimethylpyrroline N-oxide, .alpha.-(4-pyridyl-1-oxide)-N-tert-butylnitrone, TEMPO, and derivs. thereof. The I derivs. include halo derivs., bifunctional derivs., conjugates with drugs or targeting mols., dimers, and cyclodextran polymers of I. Many different disorders can be treated using these compds., including diseases or disorders of the central and peripheral nervous systems and disorders arising from ischemia, infection, inflammation, oxidn. from exposure to radiation or cytotoxic compds., as well as due to naturally occurring processes (e.g. aging). I inhibited oxidn. of LDL in plasma in vitro.

IT **3376-24-7**
RL: BIOL (Biological study)
(LDL oxidn. inhibition with, for therapeutic)

RN 3376-24-7 HCAPLUS

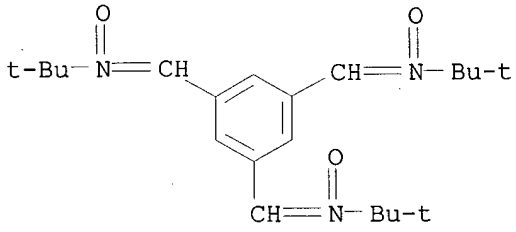
CN 2-Propanamine, 2-methyl-N-(phenylmethylene)-, N-oxide (9CI) (CA INDEX NAME)



IT **146407-39-8 146407-40-1 146407-41-2 146407-45-6**
RL: BIOL (Biological study)
(as spin trapping compd., for treatment of disease assocd. with oxidn. of lipid or protein)

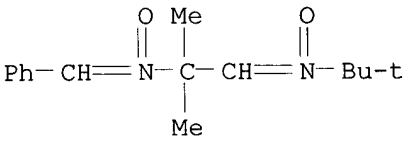
RN 146407-39-8 HCAPLUS

CN 2-Propanamine, N,N',N''-(1,3,5-benzenetriyltrimethylidyne)tris[2-methyl-, N,N',N''-trioxide (9CI) (CA INDEX NAME)



RN 146407-40-1 HCAPLUS

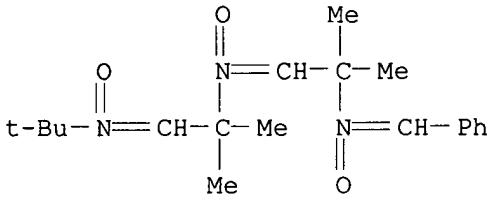
CN 2-Propanamine, 1-[(1,1-dimethylethyl)oxidoimino]-2-methyl-N-(phenylmethylene)-, N-oxide (9CI) (CA INDEX NAME)



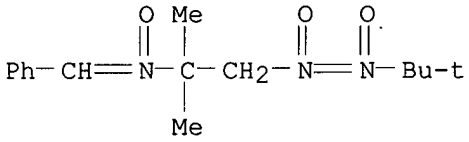
RN 146407-41-2 HCAPLUS

CN 2-Propanamine, 1-[[2-[(1,1-dimethylethyl)oxidoimino]-1,1-dimethylethyl]oxidoimino]-2-methyl-N-(phenylmethylene)-, N-oxide (9CI)

(CA INDEX NAME)



RN 146407-45-6 HCAPLUS
CN 2-Propanamine, 1-[(1,1-dimethylethyl)dioxidoazo]-2-methyl-N-(phenylmethylene)-, N-oxide (9CI) (CA INDEX NAME)



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FILE COVERS 1907 - 8 Apr 2003 VOL 138 ISS 15
FILE LAST UPDATED: 7 Apr 2003 (20030407/ED)

This file contains CAS Registry Numbers for easy and accurate substance identification.

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L13        1001 SEA FILE=HCAPLUS ABB=ON  SPIN TRAPPING+OLD/CT
L14         563 SEA FILE=HCAPLUS ABB=ON  TOXICITY+NT/CT(L)OXYGEN
L15       17178 SEA FILE=HCAPLUS ABB=ON  REACTIVE OXYGEN SPECIES/CT
L16       49447 SEA FILE=HCAPLUS ABB=ON  ANTIOXIDANTS/CT
L17       19510 SEA FILE=HCAPLUS ABB=ON  OXIDATIVE STRESS, BIOLOGICAL/CT
L18      170019 SEA FILE=HCAPLUS ABB=ON  OXIDATION/CT
L19       51371 SEA FILE=HCAPLUS ABB=ON  SKIN, DISEASE+NT/CT
L20           1 SEA FILE=REGISTRY ABB=ON  7782-44-7
L21        6845 SEA FILE=HCAPLUS ABB=ON  L20(L)ADV/RL
L25       37358 SEA FILE=HCAPLUS ABB=ON  TOPICAL?
L36           1 SEA FILE=REGISTRY ABB=ON  DMPO/CN
L37           1 SEA FILE=REGISTRY ABB=ON  POBN/CN
L38           1 SEA FILE=REGISTRY ABB=ON  TEMPO/CN
L39        2446 SEA FILE=REGISTRY ABB=ON  C11H15NO/MF
L40        1196 SEA FILE=REGISTRY ABB=ON  L39 AND 1/NR
L41           6 SEA FILE=REGISTRY ABB=ON  L40 AND NITRONE
L42           2 SEA FILE=REGISTRY ABB=ON  L41 AND 2 PROPANAMINE
L43        4040 SEA FILE=HCAPLUS ABB=ON  L36 OR L37 OR L38 OR L42
L44         400 SEA FILE=HCAPLUS ABB=ON  (L12 OR L13) AND L43
L48      135836 SEA FILE=HCAPLUS ABB=ON  (SKIN OR DERMIS OR EPIDERM? OR
                DERMAL?)/OBI
L49           2 SEA FILE=HCAPLUS ABB=ON  L44 AND ((L14 OR L15 OR L16 OR L17 OR
                L18) OR L21) AND (L19 OR L25 OR L48)
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L11       24892 SEA FILE=HCAPLUS ABB=ON  TRAPPING+OLD/CT
L12         524 SEA FILE=HCAPLUS ABB=ON  L11(L)SPIN
L13        1001 SEA FILE=HCAPLUS ABB=ON  SPIN TRAPPING+OLD/CT
L34       41883 SEA FILE=HCAPLUS ABB=ON  UV RADIATION+NT,OLD/CT
L36           1 SEA FILE=REGISTRY ABB=ON  DMPO/CN
L37           1 SEA FILE=REGISTRY ABB=ON  POBN/CN
L38           1 SEA FILE=REGISTRY ABB=ON  TEMPO/CN
L39        2446 SEA FILE=REGISTRY ABB=ON  C11H15NO/MF
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L40         1196 SEA FILE=REGISTRY ABB=ON  L39 AND 1/NR
L41            6 SEA FILE=REGISTRY ABB=ON  L40 AND NITRONE
L42            2 SEA FILE=REGISTRY ABB=ON  L41 AND 2 PROPANAMINE
L43         4040 SEA FILE=HCAPLUS ABB=ON  L36 OR L37 OR L38 OR L42
L44          400 SEA FILE=HCAPLUS ABB=ON  (L12 OR L13) AND L43
L45            2 SEA FILE=HCAPLUS ABB=ON  L34 AND L44


L11        24892 SEA FILE=HCAPLUS ABB=ON  TRAPPING+OLD/CT
L12          524 SEA FILE=HCAPLUS ABB=ON  L11(L)SPIN
L13         1001 SEA FILE=HCAPLUS ABB=ON  SPIN TRAPPING+OLD/CT
L14          563 SEA FILE=HCAPLUS ABB=ON  TOXICITY+NT/CT(L)OXYGEN
L15        17178 SEA FILE=HCAPLUS ABB=ON  REACTIVE OXYGEN SPECIES/CT
L16        49447 SEA FILE=HCAPLUS ABB=ON  ANTIOXIDANTS/CT
L17        19510 SEA FILE=HCAPLUS ABB=ON  OXIDATIVE STRESS, BIOLOGICAL/CT
L18       170019 SEA FILE=HCAPLUS ABB=ON  OXIDATION/CT
L19        51371 SEA FILE=HCAPLUS ABB=ON  SKIN, DISEASE+NT/CT
L20            1 SEA FILE=REGISTRY ABB=ON  7782-44-7
L21         6845 SEA FILE=HCAPLUS ABB=ON  L20(L)ADV/RL
L25        37358 SEA FILE=HCAPLUS ABB=ON  TOPICAL?
L36            1 SEA FILE=REGISTRY ABB=ON  DMPO/CN
L37            1 SEA FILE=REGISTRY ABB=ON  POBN/CN
L38            1 SEA FILE=REGISTRY ABB=ON  TEMPO/CN
L39         2446 SEA FILE=REGISTRY ABB=ON  C11H15NO/MF
L40         1196 SEA FILE=REGISTRY ABB=ON  L39 AND 1/NR
L41            6 SEA FILE=REGISTRY ABB=ON  L40 AND NITRONE
L42            2 SEA FILE=REGISTRY ABB=ON  L41 AND 2 PROPANAMINE
L43         4040 SEA FILE=HCAPLUS ABB=ON  L36 OR L37 OR L38 OR L42
L48       135836 SEA FILE=HCAPLUS ABB=ON  (SKIN OR DERMIS OR EPIDERM? OR
               DERMAL?)/OBI
L50          279 SEA FILE=HCAPLUS ABB=ON  L43(L)(THU OR BAC OR PAC OR PKT OR
               DMA)/RL
L51          384 SEA FILE=HCAPLUS ABB=ON  L43 AND PHARMAC?/SC,SX
L52           44 SEA FILE=HCAPLUS ABB=ON  (L50 OR L51) AND (L12 OR L13)
L53           20 SEA FILE=HCAPLUS ABB=ON  L52 AND ((L14 OR L15 OR L16 OR L17 OR
               L18 OR L19) OR L21 OR L25 OR L48)


L11        24892 SEA FILE=HCAPLUS ABB=ON  TRAPPING+OLD/CT
L12          524 SEA FILE=HCAPLUS ABB=ON  L11(L)SPIN
L13         1001 SEA FILE=HCAPLUS ABB=ON  SPIN TRAPPING+OLD/CT
L25        37358 SEA FILE=HCAPLUS ABB=ON  TOPICAL?
L99            2 SEA FILE=HCAPLUS ABB=ON  (L12 OR L13) AND L25


=> s (l49 or l45 or l53 or l99) not l108

L109          11 (L49 OR L45 OR L53 OR L99) NOT L108

=> fil uspatf; d que 165;d que 167; d que l100

FILE 'USPATFULL' ENTERED AT 11:08:49 ON 08 APR 2003
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FILE COVERS 1971 TO PATENT PUBLICATION DATE: 8 Apr 2003 (20030408/PD)
FILE LAST UPDATED: 8 Apr 2003 (20030408/ED)
HIGHEST GRANTED PATENT NUMBER: US6546558
HIGHEST APPLICATION PUBLICATION NUMBER: US2003066115
CA INDEXING IS CURRENT THROUGH 8 Apr 2003 (20030408/UPCA)
ISSUE CLASS FIELDS (/INCL) CURRENT THROUGH: 8 Apr 2003 (20030408/PD)
REVISED CLASS FIELDS (/NCL) LAST RELOADED: Feb 2003
```

previously printed

USPTO MANUAL OF CLASSIFICATIONS THESAURUS ISSUE DATE: Feb 2003

>>> USPAT2 is now available. USPATFULL contains full text of the <<<
>>> original, i.e., the earliest published granted patents or <<<
>>> applications. USPAT2 contains full text of the latest US <<<
>>> publications, starting in 2001, for the inventions covered in <<<
>>> USPATFULL. A USPATFULL record contains not only the original <<<
>>> published document but also a list of any subsequent <<<
>>> publications. The publication number, patent kind code, and <<<
>>> publication date for all the US publications for an invention <<<
>>> are displayed in the PI (Patent Information) field of USPATFULL <<<
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>>> /PK, etc. <<<

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>>> enter this cluster. <<<
>>> <<<
>>> Use USPATALL when searching terms such as patent assignees, <<<
>>> classifications, or claims, that may potentially change from <<<
>>> the earliest to the latest publication. <<<

This file contains CAS Registry Numbers for easy and accurate substance identification.

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L36           1 SEA FILE=REGISTRY ABB=ON  DMPO/CN
L37           1 SEA FILE=REGISTRY ABB=ON  POBN/CN
L38           1 SEA FILE=REGISTRY ABB=ON  TEMPO/CN
L39        2446 SEA FILE=REGISTRY ABB=ON  C11H15NO/MF
L40        1196 SEA FILE=REGISTRY ABB=ON  L39 AND 1/NR
L41           6 SEA FILE=REGISTRY ABB=ON  L40 AND NITRONE
L42           2 SEA FILE=REGISTRY ABB=ON  L41 AND 2 PROPANAMINE
L55         280 SEA FILE=USPATFULL ABB=ON  L36 OR L37 OR L38 OR L42
L56          46 SEA FILE=USPATFULL ABB=ON  (SPIN TRAP?)/IT,TI,AB,CLM
L57          23 SEA FILE=USPATFULL ABB=ON  L55 AND L56
L59       11285 SEA FILE=USPATFULL ABB=ON  TOPICAL?/IT,TI,AB,CLM
L65           7 SEA FILE=USPATFULL ABB=ON  L57 AND L59
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L36           1 SEA FILE=REGISTRY ABB=ON  DMPO/CN
L37           1 SEA FILE=REGISTRY ABB=ON  POBN/CN
L38           1 SEA FILE=REGISTRY ABB=ON  TEMPO/CN
L39        2446 SEA FILE=REGISTRY ABB=ON  C11H15NO/MF
L40        1196 SEA FILE=REGISTRY ABB=ON  L39 AND 1/NR
L41           6 SEA FILE=REGISTRY ABB=ON  L40 AND NITRONE
L42           2 SEA FILE=REGISTRY ABB=ON  L41 AND 2 PROPANAMINE
L55         280 SEA FILE=USPATFULL ABB=ON  L36 OR L37 OR L38 OR L42
L56          46 SEA FILE=USPATFULL ABB=ON  (SPIN TRAP?)/IT,TI,AB,CLM
L57          23 SEA FILE=USPATFULL ABB=ON  L55 AND L56
L58       74748 SEA FILE=USPATFULL ABB=ON  (UV OR ULTRAVIOLET OR ULTRA VIOLET
                OR SKIN OR DERMAL OR EPIDERM? OR DERMIS)/IT,TI,AB,CLM
L67           4 SEA FILE=USPATFULL ABB=ON  L57 AND L58
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L56          46 SEA FILE=USPATFULL ABB=ON  (SPIN TRAP?)/IT,TI,AB,CLM
L59       11285 SEA FILE=USPATFULL ABB=ON  TOPICAL?/IT,TI,AB,CLM
L100          9 SEA FILE=USPATFULL ABB=ON  L56 AND L59
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=> s l65 or l67 or l100

L110 11 L65 OR L67 OR L100

=> fil medl; d que l102; d que l78; d que l81; d que l84; d que l86

FILE 'MEDLINE' ENTERED AT 11:08:51 ON 08 APR 2003

FILE LAST UPDATED: 6 APR 2003 (20030406/UP). FILE COVERS 1958 TO DATE.

On June 9, 2002, MEDLINE was reloaded. See HELP RLOAD for details.

MEDLINE thesauri in the /CN, /CT, and /MN fields incorporate the MeSH 2003 vocabulary. See http://www.nlm.nih.gov/mesh/summ2003.html for a description on changes.

This file contains CAS Registry Numbers for easy and accurate substance identification.

L73 2592 SEA FILE=MEDLINE ABB=ON SPIN TRAP?
L85 31892 SEA FILE=MEDLINE ABB=ON ADMINISTRATION, TOPICAL+NT/CT
L102 2 SEA FILE=MEDLINE ABB=ON L73 AND L85

L36 1 SEA FILE=REGISTRY ABB=ON DMPO/CN
L37 1 SEA FILE=REGISTRY ABB=ON POBN/CN
L38 1 SEA FILE=REGISTRY ABB=ON TEMPO/CN
L39 2446 SEA FILE=REGISTRY ABB=ON C11H15NO/MF
L40 1196 SEA FILE=REGISTRY ABB=ON L39 AND 1/NR
L41 6 SEA FILE=REGISTRY ABB=ON L40 AND NITRONE
L42 2 SEA FILE=REGISTRY ABB=ON L41 AND 2 PROPANAMINE
L72 1114 SEA FILE=MEDLINE ABB=ON L36 OR L37 OR L38 OR L42
L73 2592 SEA FILE=MEDLINE ABB=ON SPIN TRAP?
L74 669 SEA FILE=MEDLINE ABB=ON L72 AND L73
L75 393433 SEA FILE=MEDLINE ABB=ON SKIN DISEASES+NT/CT
L78 3 SEA FILE=MEDLINE ABB=ON L74 AND L75

L36 1 SEA FILE=REGISTRY ABB=ON DMPO/CN
L37 1 SEA FILE=REGISTRY ABB=ON POBN/CN
L38 1 SEA FILE=REGISTRY ABB=ON TEMPO/CN
L39 2446 SEA FILE=REGISTRY ABB=ON C11H15NO/MF
L40 1196 SEA FILE=REGISTRY ABB=ON L39 AND 1/NR
L41 6 SEA FILE=REGISTRY ABB=ON L40 AND NITRONE
L42 2 SEA FILE=REGISTRY ABB=ON L41 AND 2 PROPANAMINE
L72 1114 SEA FILE=MEDLINE ABB=ON L36 OR L37 OR L38 OR L42
L73 2592 SEA FILE=MEDLINE ABB=ON SPIN TRAP?
L74 669 SEA FILE=MEDLINE ABB=ON L72 AND L73
L76 13000 SEA FILE=MEDLINE ABB=ON OXIDATIVE STRESS/CT
L80 119296 SEA FILE=MEDLINE ABB=ON SKIN+NT/CT
L81 2 SEA FILE=MEDLINE ABB=ON L74 AND L76 AND L80

L36 1 SEA FILE=REGISTRY ABB=ON DMPO/CN
L37 1 SEA FILE=REGISTRY ABB=ON POBN/CN
L38 1 SEA FILE=REGISTRY ABB=ON TEMPO/CN
L39 2446 SEA FILE=REGISTRY ABB=ON C11H15NO/MF
L40 1196 SEA FILE=REGISTRY ABB=ON L39 AND 1/NR
L41 6 SEA FILE=REGISTRY ABB=ON L40 AND NITRONE
L42 2 SEA FILE=REGISTRY ABB=ON L41 AND 2 PROPANAMINE

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L72         1114 SEA FILE=MEDLINE ABB=ON  L36 OR L37 OR L38 OR L42
L73         2592 SEA FILE=MEDLINE ABB=ON  SPIN TRAP?
L74          669 SEA FILE=MEDLINE ABB=ON  L72 AND L73
L76        13000 SEA FILE=MEDLINE ABB=ON  OXIDATIVE STRESS/CT
L82         2588 SEA FILE=MEDLINE ABB=ON  L76(L)DE/CT
L84            5 SEA FILE=MEDLINE ABB=ON  L82/MAJ AND L74
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-Subheading DE = drug effects

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L36            1 SEA FILE=REGISTRY ABB=ON  DMPO/CN
L37            1 SEA FILE=REGISTRY ABB=ON  POBN/CN
L38            1 SEA FILE=REGISTRY ABB=ON  TEMPO/CN
L39         2446 SEA FILE=REGISTRY ABB=ON  C11H15NO/MF
L40         1196 SEA FILE=REGISTRY ABB=ON  L39 AND 1/NR
L41            6 SEA FILE=REGISTRY ABB=ON  L40 AND NITRONE
L42            2 SEA FILE=REGISTRY ABB=ON  L41 AND 2 PROPANAMINE
L72         1114 SEA FILE=MEDLINE ABB=ON  L36 OR L37 OR L38 OR L42
L73         2592 SEA FILE=MEDLINE ABB=ON  SPIN TRAP?
L74          669 SEA FILE=MEDLINE ABB=ON  L72 AND L73
L85        31892 SEA FILE=MEDLINE ABB=ON  ADMINISTRATION, TOPICAL+NT/CT
L86            0 SEA FILE=MEDLINE ABB=ON  L74 AND L85

=> s l102 or l78 or l81 or l84

L111          11 L102 OR L78 OR L81 OR L84

=> fil embase

FILE 'EMBASE' ENTERED AT 11:08:53 ON 08 APR 2003
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 FILE COVERS 1974 TO 3 Apr 2003 (20030403/ED)

 EMBASE has been reloaded. Enter HELP RLOAD for details.

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 substance identification.

=> d que l93; d que l95; d que l97; d que l104; d que l107

L36            1 SEA FILE=REGISTRY ABB=ON  DMPO/CN
L37            1 SEA FILE=REGISTRY ABB=ON  POBN/CN
L38            1 SEA FILE=REGISTRY ABB=ON  TEMPO/CN
L39         2446 SEA FILE=REGISTRY ABB=ON  C11H15NO/MF
L40         1196 SEA FILE=REGISTRY ABB=ON  L39 AND 1/NR
L41            6 SEA FILE=REGISTRY ABB=ON  L40 AND NITRONE
L42            2 SEA FILE=REGISTRY ABB=ON  L41 AND 2 PROPANAMINE
L87          893 SEA FILE=EMBASE ABB=ON  L36 OR L37 OR L38 OR L42
L91         1414 SEA FILE=EMBASE ABB=ON  TOPICAL AGENT/CT
L92        71000 SEA FILE=EMBASE ABB=ON  TOPICAL DRUG ADMINISTRATION/CT
L93            5 SEA FILE=EMBASE ABB=ON  L87 AND (L91 OR L92)


L36            1 SEA FILE=REGISTRY ABB=ON  DMPO/CN
L37            1 SEA FILE=REGISTRY ABB=ON  POBN/CN
L38            1 SEA FILE=REGISTRY ABB=ON  TEMPO/CN
L39         2446 SEA FILE=REGISTRY ABB=ON  C11H15NO/MF
L40         1196 SEA FILE=REGISTRY ABB=ON  L39 AND 1/NR
L41            6 SEA FILE=REGISTRY ABB=ON  L40 AND NITRONE
L42            2 SEA FILE=REGISTRY ABB=ON  L41 AND 2 PROPANAMINE
L87          893 SEA FILE=EMBASE ABB=ON  L36 OR L37 OR L38 OR L42
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L88      364060 SEA FILE=EMBASE ABB=ON  SKIN DISEASE+NT/CT
L89       78471 SEA FILE=EMBASE ABB=ON  SKIN+NT/CT
L90       22980 SEA FILE=EMBASE ABB=ON  OXIDATIVE STRESS/CT
L95           4 SEA FILE=EMBASE ABB=ON  L87 AND (L88 OR L89) AND L90


L36           1 SEA FILE=REGISTRY ABB=ON  DMPO/CN
L37           1 SEA FILE=REGISTRY ABB=ON  POBN/CN
L38           1 SEA FILE=REGISTRY ABB=ON  TEMPO/CN
L39        2446 SEA FILE=REGISTRY ABB=ON  C11H15NO/MF
L40        1196 SEA FILE=REGISTRY ABB=ON  L39 AND 1/NR
L41           6 SEA FILE=REGISTRY ABB=ON  L40 AND NITRONE
L42           2 SEA FILE=REGISTRY ABB=ON  L41 AND 2 PROPANAMINE
L87         893 SEA FILE=EMBASE ABB=ON  L36 OR L37 OR L38 OR L42
L88      364060 SEA FILE=EMBASE ABB=ON  SKIN DISEASE+NT/CT
L89       78471 SEA FILE=EMBASE ABB=ON  SKIN+NT/CT
L90       22980 SEA FILE=EMBASE ABB=ON  OXIDATIVE STRESS/CT
L94       20578 SEA FILE=EMBASE ABB=ON  ULTRAVIOLET RADIATION/CT
L97           6 SEA FILE=EMBASE ABB=ON  L87 AND L94 AND ((L88 OR L89 OR L90))


L91        1414 SEA FILE=EMBASE ABB=ON  TOPICAL AGENT/CT
L92       71000 SEA FILE=EMBASE ABB=ON  TOPICAL DRUG ADMINISTRATION/CT
L103       2416 SEA FILE=EMBASE ABB=ON  SPIN TRAP?
L104          6 SEA FILE=EMBASE ABB=ON  L103 AND (L91 OR L92)


L36           1 SEA FILE=REGISTRY ABB=ON  DMPO/CN
L37           1 SEA FILE=REGISTRY ABB=ON  POBN/CN
L38           1 SEA FILE=REGISTRY ABB=ON  TEMPO/CN
L39        2446 SEA FILE=REGISTRY ABB=ON  C11H15NO/MF
L40        1196 SEA FILE=REGISTRY ABB=ON  L39 AND 1/NR
L41           6 SEA FILE=REGISTRY ABB=ON  L40 AND NITRONE
L42           2 SEA FILE=REGISTRY ABB=ON  L41 AND 2 PROPANAMINE
L87         893 SEA FILE=EMBASE ABB=ON  L36 OR L37 OR L38 OR L42
L88      364060 SEA FILE=EMBASE ABB=ON  SKIN DISEASE+NT/CT
L89       78471 SEA FILE=EMBASE ABB=ON  SKIN+NT/CT
L90       22980 SEA FILE=EMBASE ABB=ON  OXIDATIVE STRESS/CT
L94       20578 SEA FILE=EMBASE ABB=ON  ULTRAVIOLET RADIATION/CT
L103       2416 SEA FILE=EMBASE ABB=ON  SPIN TRAP?
L105        590 SEA FILE=EMBASE ABB=ON  L87 AND L103
L107          4 SEA FILE=EMBASE ABB=ON  L105 AND (L88 OR L89) AND (L90 OR L94)


=> s l93 or l95 or l97 or l104 or l107

L112         15 L93 OR L95 OR L97 OR L104 OR L107

=> dup rem l109,l110,l111,l112
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FILE 'MEDLINE' ENTERED AT 11:09:21 ON 08 APR 2003
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FILE 'EMBASE' ENTERED AT 11:09:21 ON 08 APR 2003
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PROCESSING COMPLETED FOR L109
PROCESSING COMPLETED FOR L110
PROCESSING COMPLETED FOR L111
PROCESSING COMPLETED FOR L112
L113 44 DUP REM L109 L110 L111 L112 (4 DUPLICATES REMOVED)
ANSWERS '1-11' FROM FILE HCAPLUS
ANSWERS '12-21' FROM FILE USPATFULL
ANSWERS '22-32' FROM FILE MEDLINE
ANSWERS '33-44' FROM FILE EMBASE

=> d ibib ab hitrn 1-21; d iall 22-44

L113 ANSWER 1 OF 44 HCAPLUS COPYRIGHT 2003 ACS DUPLICATE 1
ACCESSION NUMBER: 1997:701470 HCAPLUS
DOCUMENT NUMBER: 128:7308
TITLE: DMPO spin-trapping compositions and methods of use thereof
INVENTOR(S): Janzen, Edward G.; Zhang, Yong-kang
PATENT ASSIGNEE(S): Oklahoma Medical Research Foundation, USA
SOURCE: U.S., 14 pp., Cont.-in-part of U.S. Ser. No. 716,952, abandoned.
CODEN: USXXAM
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 4
PATENT INFORMATION:

| PATENT NO. | KIND | DATE | APPLICATION NO. | DATE |
|---|---|---|---|---|
| US 5681845 | A | 19971028 | US 1993-11968 | 19930201 |
| US 5025032 | A | 19910618 | US 1989-422651 | 19891017 |
| JP 09025263 | A2 | 19970128 | JP 1996-179709 | 19901017 |
| JP 10259128 | A2 | 19980929 | JP 1998-77985 | 19901017 |
| JP 2002179563 | A2 | 20020626 | JP 2001-343709 | 19901017 |
| US 5622994 | A | 19970422 | US 1994-212800 | 19940315 |
| US 5578617 | A | 19961126 | US 1994-365548 | 19941228 |
| US 5681965 | A | 19971028 | US 1995-468561 | 19950606 |
| US 6107315 | A | 20000822 | US 1995-468563 | 19950606 |
| US 6002001 | A | 19991214 | US 1997-969344 | 19971128 |
| JP 10259178 | A2 | 19980929 | JP 1998-77984 | 19980325 |
| JP 2975587 | B2 | 19991110 | | |
| AU 9883101 | A1 | 19981224 | AU 1998-83101 | 19980904 |
| AU 710341 | B2 | 19990916 | | |
| US 6403627 | B1 | 20020611 | US 1999-357297 | 19990720 |

PRIORITY APPLN. INFO.:
US 1989-422651 A2 19891017
US 1990-589177 B2 19900927
US 1991-716952 B2 19910618
JP 1990-515036 A3 19901017
JP 1996-179709 A3 19901017
JP 1998-77985 A3 19901017
US 1993-27559 A3 19930305
US 1993-52870 B1 19930426
US 1994-212800 A2 19940315
US 1994-167900 B1 19940729
US 1994-365548 A1 19941228
AU 1995-11315 A3 19950120
US 1997-969344 A1 19971128

OTHER SOURCE(S): MARPAT 128:7308
AB Spin-trapping compns. in general have now been discovered to be effective in treating a variety of disorders, including disorders such as those arising from ischemia, infection, inflammation, exposure to radiation or

cytotoxic compds., not just of the central and peripheral nervous systems but of peripheral organ disease having a wide variety of etiologies. In the preferred embodiment, the compns. for treating tissue damage from ischemia include 5,5-dimethylpyrroline N-oxide (DMPO), and spin-trapping derivs. thereof, in a suitable pharmaceutical carrier for i.v., oral, **topical**, or nasal/pulmonary administration. Many different disorders can be treated using these compds., including diseases or disorders of the central and peripheral nervous systems, and disorders arising from ischemia, infection, inflammation, oxidn. from exposure to radiation or cytotoxic compds., as well as due to naturally occurring processes such as aging.

IT **3317-61-1**, Dmpo
RL: **BAC (Biological activity or effector, except adverse)**; BSU (Biological study, unclassified); PEP (Physical, engineering or chemical process); **THU (Therapeutic use)**; BIOL (Biological study); PROC (Process); USES (Uses)
(DMPO spin-trapping compns. and methods of use thereof for treatment of ischemia, infection, inflammation, and aging)

L113 ANSWER 2 OF 44 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2002:911629 HCAPLUS
DOCUMENT NUMBER: 138:204638
TITLE: Effects of resveratrol and its analogs on scavenging hydroxyl radicals: evaluation by EPR spin trapping method
AUTHOR(S): Lu, M.; Fang, J.-G.; Liu, Z.-L.; Wu, L.-M.
CORPORATE SOURCE: National Laboratory of Applied Organic Chemistry, Lanzhou University, Lanzhou, ~~Peop.~~ Rep. China
SOURCE: Applied Magnetic Resonance (2002), 22(4), 475-481
CODEN: APMREI; ISSN: 0937-9347
PUBLISHER: Springer-Verlag Wien
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Resveratrol (3,4',5-trihydroxy-trans-stilbene) and six analogs, polyhydroxystilbenes, were synthesized. Their effects on scavenging hydroxyl radicals were studied by ESR spin trapping method. The EPR signal intensity of the spin adduct of hydroxyl radical to 5,5-dimethyl-1-pyrroline N-oxide was detected and used as a std. for the evaluation of the effect of the seven compds. on scavenging hydroxyl radicals. While all seven compds. exhibited hydroxyl radical-scavenging activity, some of them proved to be more effective than resveratrol in this model. Another stable but low-intensity spin adduct was also obsd. by EPR. A possible assignment is proposed.

IT **3317-61-1**, DMPO
RL: ARG (Analytical reagent use); RCT (Reactant); ANST (Analytical study); RACT (Reactant or reagent); USES (Uses)
(spin trapping agent; ESR spin trapping study on the activity of resveratrol and its analogs in scavenging hydroxyl radicals)

REFERENCE COUNT: 13 THERE ARE 13 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L113 ANSWER 3 OF 44 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2002:576831 HCAPLUS
DOCUMENT NUMBER: 137:165680
TITLE: Sterilization system using microwave and UV light
AUTHOR(S): Iwaguch, Shiro; Matsumura, Kentaro; Tokuoka, Yoshikazu; Wakui, Shiro; Kawashima, Norimichi
CORPORATE SOURCE: Faculty of Engineering, Toin University of Yokohama, Yokohama, 225-8502, Japan
SOURCE: Colloids and Surfaces, B: Biointerfaces (2002), 25(4), 299-304
CODEN: CSBBEQ; ISSN: 0927-7765
PUBLISHER: Elsevier Science B.V.

Searched by Barb O'Bryen, STIC 308-4291

DOCUMENT TYPE: Journal
LANGUAGE: English
AB We constructed a novel microwave-UV light sterilization system and investigated its sterilization effect. This sterilization system can emit UV light by irradn. of microwave without other power. When irradiating UV light with and/or without microwave on aq. DMPO soln., active oxygen species such as hydroxyl radical or superoxide were generated in the soln. The amt. of active oxygen species generated by irradn. of microwave and UV light was larger than that by irradn. of UV light alone. This would be due to the promotion of emission of UV light photons by microwave and UV light irradn. Moreover, microwave-UV light sterilization was highly effective to sterilize microorganisms. The generation of active oxygen species would play an important role in sterilization of the sterilization system.
IT **3317-61-1**, DMPO
RL: ARU (Analytical role, unclassified); ANST (Analytical study)
(sterilization system using microwave and UV light)
REFERENCE COUNT: 8 THERE ARE 8 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L113 ANSWER 4 OF 44 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2001:545474 HCAPLUS
DOCUMENT NUMBER: 135:117264
TITLE: Free radical scavengers or promoters thereof as therapeutic adjuvants in preterm parturition
INVENTOR(S): Buhimschi, Irina; Weiner, Carl P.
PATENT ASSIGNEE(S): USA
SOURCE: PCT Int. Appl., 49 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

| PATENT NO. | KIND | DATE | APPLICATION NO. | DATE |
|---|---|---|---|---|
| WO 2001052835 | A2 | 20010726 | WO 2001-US1710 | 20010118 |
| WO 2001052835 | A3 | 20011220 | | |
| W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM | | | | |
| RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG | | | | |
| US 2001031731 | A1 | 20011018 | US 2001-765476 | 20010118 |
| EP 1263428 | A2 | 20021211 | EP 2001-904920 | 20010118 |
| R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR | | | | |

PRIORITY APPLN. INFO.: US 2000-176575P P 20000118
WO 2001-US1710 W 20010118

AB The usage of compds. that improve fetal and neonatal outcome of preterm birth is described. These compds. are scavengers of reactive oxygen species (ROS), their precursors, and agents that induce prodn. of the scavengers. Examples of these compds. are glutathione, N-acetylcysteine, antioxidants, and spin trapping compds. These compds. improve fetal outcome by inhibiting a fetal inflammatory process that may affect the fetus independently of prematurity. This fetal inflammatory response is characterized by increased cytokine and matrix metalloproteases (MMP) levels both in the mother and fetus and may be modulated by ROS at different levels. Targeting ROS formation with compds. such as specific

antioxidants, glutathione or spin trapping compds., their precursors, and/or agents which induce their prodn. will suppress both the direct effects of ROS and its indirect effects through cytokines and MMPs already circulating in the system. This therapeutic intervention would limit the pathophysiol. chain of events that ultimately leads to preterm premature rupture of membranes (PPROM), preterm birth and/or adverse fetal and neonatal outcome.

IT **7782-44-7D**, Oxygen, reactive species
RL: **ADV (Adverse effect, including toxicity)**; BAC (Biological activity or effector, except adverse); BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(free radical scavengers as therapeutic adjuvants in preterm parturition)

IT **66893-81-0D**, POBN, hydroxyl radical adducts
RL: BOC (Biological occurrence); BSU (Biological study, unclassified); BIOL (Biological study); OCCU (Occurrence)
(free radical scavengers as therapeutic adjuvants in preterm parturition)

IT **66893-81-0**, POBN
RL: **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(free radical scavengers as therapeutic adjuvants in preterm parturition)

L113 ANSWER 5 OF 44 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2001:670657 HCAPLUS
DOCUMENT NUMBER: 136:564
TITLE: Spin-trapping study on the hydroxyl radical formed from a tea catechin-Cu(II) system
AUTHOR(S): Yoshioka, Hisashi; Senba, Yasushi; Saito, Kieko; Kimura, Takahide; Hayakawa, Fumiko
CORPORATE SOURCE: Institute for Environmental Sciences, University of Shizuoka, Shizuoka, 422-8529, Japan
SOURCE: Bioscience, Biotechnology, and Biochemistry (2001), 65(8), 1697-1706
CODEN: BBBIEJ; ISSN: 0916-8451
PUBLISHER: Japan Society for Bioscience, Biotechnology, and Agrochemistry
DOCUMENT TYPE: Journal
LANGUAGE: English

AB A spin-trapping method was applied to examine the formation of the hydroxyl (OH) radical from a tea catechin-Cu(II) system to elucidate a previous result that some tea catechin-Cu(II) systems induced DNA scission. Three tea catechins, (-)-epigallocatechin (EGC), (-)-epigallocatechin gallate (EGCg), and (-)-epicatechin (EC), were used. The spin-trapping agent, 5,5'-dimethyl-pyrroline-1-oxide (DMPO), was dissolved in a pH 9 phosphate buffer soln., then a catechin and Cu(II) were added in that order, and the ESR spectral change was monitored for 1 h. The order of adding the catechin and Cu(II) was then reversed, and the ESR spectral change was again monitored to examine the coordinating activity of each catechin toward the Cu(II) ion and the effect on OH radical generation. The intensity changes of the spin adducts, DMPO-OH, DMPO-CH3, and DMPO-H, were analyzed, the results suggesting that the OH radical generated in the system decompd. DMPO, resulting in the formation of DMPO-CH3 and DMPO-H. The results show that EGC formed a stable complex with Cu(II) and generated the OH radical. EGCg seemed to have this activity, but the OH radical that was generated was scavenged by the gallate group existing in the complex. EC did not show strong coordinating and OH-generating activities. These characteristics of the 3 catechins are consistent with the results shown for DNA scission.

IT **3317-61-1**, DMPO
RL: ARG (Analytical reagent use); ANST (Analytical study); USES (Uses)
(spin-trapping study on the OH radical formed from a tea catechin-Cu(II) system)

REFERENCE COUNT: 33 THERE ARE 33 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L113 ANSWER 6 OF 44 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2001:220080 HCAPLUS
DOCUMENT NUMBER: 135:56021
TITLE: Spin trapping agents (tempol and POBN) protect HepG2 cells overexpressing CYP2E1 against arachidonic acid toxicity
AUTHOR(S): Perez, M. J.; Cederbaum, A. I.
CORPORATE SOURCE: Department of Biochemistry and Molecular Biology, Mount Sinai School of Medicine, New York, NY, USA
SOURCE: Free Radical Biology & Medicine (2001), 30(7), 734-746
CODEN: FRBMEH; ISSN: 0891-5849
PUBLISHER: Elsevier Science Inc.
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Polyunsatd. fatty acids such as arachidonic acid were previously shown to be toxic to HepG2 cells expressing CYP2E1 by a mechanism involving oxidative stress and lipid peroxidn. This study investigated the effects of the spin trapping agents Tempol and POBN on the arachidonic acid toxicity. Arachidonic acid caused toxicity and induced lipid peroxidn. and mitochondrial membrane damage in cells overexpressing CYP2E1 but had little or no effect in control cells not expressing CYP2E1. The toxicity appeared to be both apoptotic and necrotic in nature. 4-Hydroxy-[2,2,6,6-tetramethylpiperidine-1-oxyl] (Tempol) and .alpha.-(4-pyridyl-1-oxide)-N-tert-Bu nitrone (POBN) protected against the decrease in cell viability and the apoptosis and necrosis. These spin traps prevented the enhanced lipid peroxidn. and the loss of mitochondrial membrane potential. Tempol and POBN had little or no effect on cellular viability or on CYP2E1 activity at concns. which were protective. It is proposed that elevated prodn. of reactive oxygen intermediates by cells expressing CYP2E1 can cause lipid peroxidn., which subsequently damages the mitochondrial membrane leading to a loss in cell viability when the cells are enriched with arachidonic acid. Tempol and POBN, which scavenge various radical intermediates, prevent in this way the enhanced lipid peroxidn., mitochondrial dysfunction, and the cell toxicity. Since oxidative stress appears to play a key role in ethanol hepatotoxicity, it may be of interest to evaluate whether such spin trapping agents are useful candidates for the prevention or improvement of ethanol-induced liver injury.

IT **66893-81-0**, POBN
RL: **BAC (Biological activity or effector, except adverse)**; BSU (Biological study, unclassified); BIOL (Biological study)
(spin trapping agents protect HepG2 cells overexpressing CYP2E1 against arachidonic acid toxicity)

REFERENCE COUNT: 82 THERE ARE 82 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L113 ANSWER 7 OF 44 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2001:240269 HCAPLUS
DOCUMENT NUMBER: 135:87124
TITLE: Superoxide scavenging activities of sixty chinese medicines determined by an ESR spin-trapping method using electrogenerated superoxide
AUTHOR(S): Liu, Wenwei; Ogata, Tateaki; Sato, Shigeyoshi; Unoura, Kei; Onodera, Jun-ichi
CORPORATE SOURCE: Graduate School of Science and Engineering, Yamagata University, Yonezawa, 992-8510, Japan
SOURCE: Yakugaku Zasshi (2001), 121(4), 265-270
CODEN: YKKZAJ; ISSN: 0031-6903
PUBLISHER: Pharmaceutical Society of Japan
DOCUMENT TYPE: Journal

LANGUAGE: Japanese
AB Superoxide-scavenging activities of 60 kinds of Chinese herbal medicines were detd. accurately by an ESR (ESR) spin-trapping technique using 5,5-dimethyl-1-pyrroline 1-oxide (DMPO) as a spin-trapping reagent. As a source of superoxide in this method, superoxide generated by one-electron redn. of the oxygen mol. in DMSO soln. was used. As a result of these studies, very powerful scavenging activity was found in Chinese medicines for inflammation, diseases of blood circulation and for tumors.
IT **3317-61-1**, 5,5-Dimethyl-1-pyrroline 1-oxide
RL: ARG (Analytical reagent use); ANST (Analytical study); USES (Uses)
(superoxide scavenging activities of sixty chinese medicines detd. by ESR spin-trapping method using electrogenerated superoxide)

L113 ANSWER 8 OF 44 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2000:423547 HCAPLUS
DOCUMENT NUMBER: 133:89159
TITLE: A new method for measuring scavenging activity of antioxidants to the hydroxyl radical formed by gamma-irradiation
AUTHOR(S): Yoshioka, Hiroe; Ohashi, Yasunori; Akaboshi, Mitsuhiko; Yoshioka, Hisashi
CORPORATE SOURCE: Radiochemistry Research Laboratory, Faculty of Science, Shizuoka University, Shizuoka, 422- 8529, Japan
SOURCE: JAERI-Conf (2000), 2000-001(JCBSRC '99, the 8th Japan-China Bilateral Symposium on Radiation Chemistry, 1999), 33-37
CODEN: JECNEC
PUBLISHER: Japan Atomic Energy Research Institute
DOCUMENT TYPE: Journal
LANGUAGE: English
AB A conference. A new method using ESR spin trapping was proposed for measuring scavenging activity of antioxidants to the hydroxyl (OH) radical. (-)-Epigallocatechin gallate (EGCg) and 5,5-dimethyl-1-pyrroline N-oxide (DMPO) were used as an antioxidant and a spin trapping agent, resp. Conventional method using a Fenton reaction had some defects on the estn. of the activity, because antioxidant disturbed the generating system of OH radical besides it scavenged the spin adduct (DMPO-OH). This method used intense .gamma.-irradn. as OH radical generating system, and the intensity decrease of DMPO-OH after the end of the irradn. was followed to obtain the rate const. of the scavenging of DMPO-OH with EGCg and to est. the quantity of DMPO-OH formed during .gamma.-irradn. By using these values, the reaction rate const. between OH radical and EGCg was calcd. as a ratio to that of DMPO. This method is useful to compare precisely the OH radical scavenging activity of various antioxidants.
IT **3317-61-1**, 5,5-Dimethyl-1-pyrroline N-oxide
RL: PEP (Physical, engineering or chemical process); PRP (Properties); RCT (Reactant); PROC (Process); RACT (Reactant or reagent)
(spin trap; method for detg. scavenging activity of antioxidants to hydroxyl radical formed by gamma-irradn.)
REFERENCE COUNT: 14 THERE ARE 14 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L113 ANSWER 9 OF 44 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 1999:691865 HCAPLUS
DOCUMENT NUMBER: 132:22813
TITLE: Electron paramagnetic resonance and spectrophotometric evidence on the photodynamic activity of a new perylenequinonoid pigment
AUTHOR(S): He, Yu-Ying; An, Jing-Yi; Jiang, Li-Jin
CORPORATE SOURCE: Institute of Photographic Chemistry, Academia Sinica, Beijing, 100101, Peop. Rep. China
SOURCE: Journal of Photochemistry and Photobiology, B: Biology

(1999), 50(2-3), 166-173
CODEN: JPPBEG; ISSN: 1011-1344
PUBLISHER: Elsevier Science S.A.
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Di-cysteine substituted hypocrellin B (DCHB) is a new water-sol. photosensitizer with significantly enhanced red absorption at wavelengths longer than 600 nm over the parent compd. hypocrellin B (HB). The photosensitizing properties (Type I and/or Type II mechanisms) of DCHB have been investigated in dimethylsulfoxide (DMSO) and aq. soln. (pH 7.4) using ESR and spectrophotometric methods. In anaerobic DMSO soln., the semiquinone anion radical of DCHB (DCHB.cntdot.-) is predominantly photoproduced via self-electron transfer between excited- and ground-state DCHB species. The presence of an electron donor significantly promotes the formation of the reduced form of DCHB. When a deoxygenated aq. soln. of DCHB and an electron donor are irradiated with 532 nm light, the hydroquinone of DCHB (DCHBH2) is formed via the disproportionation of the first-formed DCHB.cntdot.- and second electron transfer to DCHB.cntdot.- from the electron donor. When oxygen is present, singlet oxygen (1O2), superoxide anion radical (O2.cntdot.-) and hydroxyl radical (.cntdot.OH) are produced. The quantum yield of 1O2 generation by DCHB photosensitization is estd. to be 0.54 using Rose Bengal as a ref., a little lower than that of HB (0.76). The superoxide anion radical is also significantly enhanced by the presence of electron donors. Moreover, O2.cntdot.- upon disproportionation generated H2O2 and ultimately the highly reactive .cntdot.OH via the Haber-Weiss reaction pathway. The efficiency of O2.cntdot.- generation by DCHB is obviously enhanced over that of HB. These findings suggest that the photodynamic actions of DCHB may proceed via Type I and Type II mechanisms and that this new photosensitizer retains photosensitizing activity after photodynamic therapy-oriented chem. modification.

IT **2564-83-2**, TEMPO
RL: FMU (Formation, unclassified); PRP (Properties); FORM (Formation, nonpreparative)
(ESR and spectrophotometric evidence of photodynamic activity of a perylenequinonoid pigment for the formation of reactive oxygen species superoxide, hydroxyl, and singlet oxygen)

IT **3317-61-1**, DMPO
RL: ARG (Analytical reagent use); RCT (Reactant); ANST (Analytical study); RACT (Reactant or reagent); USES (Uses)
(spin trap for superoxide and hydroxyl; ESR and spectrophotometric evidence of photodynamic activity of a perylenequinonoid pigment for the formation of reactive oxygen species superoxide, hydroxyl, and singlet oxygen)

REFERENCE COUNT: 36 THERE ARE 36 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L113 ANSWER 10 OF 44 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 1997:477648 HCAPLUS
DOCUMENT NUMBER: 127:156677
TITLE: Use of spin-traps during warm ischemia-reperfusion in rat liver: comparative effect on energetic metabolism studied using 31P nuclear magnetic resonance
AUTHOR(S): Delmas-Beauvieux, M.C.; Pietri, S.; Culcasi, M.; Leducq, N.; Valeins, H.; Liebgott, T.; Diolez, P.; Canioni, P.; Gallis, J.L.
CORPORATE SOURCE: Laboratoire de Resonance Magnetique des Systemes Biologiques, Universite Victor Segalen Bordeaux 2, Bordeaux, F-33076, Fr.
SOURCE: Magnetic Resonance Materials in Physics, Biology, and Medicine (1997), 5(1), 45-52
CODEN: MRBMEQ; ISSN: 1352-8661
PUBLISHER: Chapman & Hall

DOCUMENT TYPE: Journal
LANGUAGE: English
AB Detection of free radicals by ESR (ESR) proves the involvement of reactive oxygen species (ROS) in reperfused organ injuries. Spin-traps are known to ameliorate hemodynamic parameters in an isolated postischemic heart. The effects of 5 nmol/L DMPO (5,5-dimethyl-1-pyrroline-N-oxide) or DEPMPO (5-(diethoxyphosphoryl)-5-methyl-1-pyrroline N-oxide) on intracellular pH (pHin) and ATP level were evaluated by 31P NMR on isolated rat liver submitted to 1 h of warm ischemia and reperfusion. At the end of the reperfusion period, during which pHin recovered to its initial value (7.16 .+-. 0.03) in all groups, the ATP recovery level (expressed in percentage of initial value) was similar in controls and DEPMPO (60% .+-. 5%, n = 6 and 54% .+-. 4%, n = 6, resp.), but only 37% .+-. 1% in DMPO-treated livers (n = 6) ($p < 0.05$ vs. controls and $p < 0.05$ vs. DEPMPO). Oxidative phosphorylation was not affected by an addn. of nitrones on isolated mitochondria extd. from livers not submitted to ischemia-reperfusion. In contrast, mitochondria extd. at the end of the ischemia-reperfusion showed an impairment in the phosphorylation parameters, particularly in the presence of DMPO. Mass spectrum of ischemic liver perchloric acid exts. evidenced probable catabolites in treated groups. The differences in the effect of the two nitrones on energetic metab. may be explained by the prodn. of deleterious catabolites by DMPO as compared to DEPMPO. Even though a specific radical scavenging effect could be operative in the liver, our results indicate that catabolic effects were predominant. The absence of deleterious effects of DEPMPO in contrast to DMPO on the liver energetic metab. was evidenced, allowing the use of DEPMPO for ESR detection.
IT **3317-61-1**, DMPO
RL: ADV (Adverse effect, including toxicity); **BAC (Biological activity or effector, except adverse)**; BSU (Biological study, unclassified); BIOL (Biological study)
(spin-trap effect on energy metab. during warm ischemia-reperfusion in rat liver)

L113 ANSWER 11 OF 44 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 1992:525965 HCAPLUS
DOCUMENT NUMBER: 117:125965
TITLE: Stabilities of hydroxyl radical spin adducts of PBN-type spin traps
AUTHOR(S): Janzen, Edward G.; Kotake, Yashige; Hinton, Randall D.
CORPORATE SOURCE: Natl. Biomed. Cent. Spin Trapping, Oklahoma Med. Res. Found., Oklahoma City, OK, 73104, USA
SOURCE: Free Radical Biology & Medicine (1992), 12(2), 169-73
CODEN: FRBMEH; ISSN: 0891-5849
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The stability of the hydroxyl spin adduct of nine different PBN-type spin traps was examd. in phosphate buffer solns. of various pH. The hydroxyl adduct is produced by short illumination of hydrogen peroxide with UV light in the presence of spin trap and the decay of its EPR signal followed. The stability measured by the half life of the first-order decay is strongly dependent on the pH of the soln. and the structure of the arom. ring used in the trap. All hydroxyl adducts are more stable in acidic media. tert-Bu hydroaminoxyl is detected as a degrdn. product of the hydroxyl adduct from all spin traps.
IT **3376-24-7 66893-81-0**
RL: PRP (Properties)
(stability of, after hydrogen peroxide reaction with spin trap, pH effect on, hydroxyl radical detection by EPR in relation to)

L113 ANSWER 12 OF 44 USPATFULL
ACCESSION NUMBER: 2002:276124 USPATFULL
TITLE: Methods for in vivo reduction of free radical levels

and compositions useful therefor
INVENTOR(S): Lai, Ching-San, Encinitas, CA, United States
PATENT ASSIGNEE(S): MCW Research Foundation, Inc., Milwaukee, WI, United States (U.S. corporation)

| | NUMBER | KIND | DATE |
|---|---|---|---|
| PATENT INFORMATION: | US 6469057 | B1 | 20021022 |
| APPLICATION INFO.: | US 2000-672140 | | 20000927 (9) |

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 1997-863059, filed on 23 May 1997, now abandoned Continuation-in-part of Ser. No. US 1996-767125, filed on 9 Dec 1996, now patented, Pat. No. US 5847004, issued on 8 Dec 1998 Continuation-in-part of Ser. No. US 1995-554196, filed on 6 Nov 1995, now patented, Pat. No. US 5741815, issued on 21 Apr 1998 Continuation-in-part of Ser. No. US 1995-459518, filed on 2 Jun 1995, now patented, Pat. No. US 5756540, issued on 26 May 1998
DOCUMENT TYPE: Utility
FILE SEGMENT: GRANTED
PRIMARY EXAMINER: Aulakh, Charanjit S.
LEGAL REPRESENTATIVE: Reiter, Stephen E., Foley & Lardner
NUMBER OF CLAIMS: 42
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 13 Drawing Figure(s); 6 Drawing Page(s)
LINE COUNT: 1380
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB In accordance with the present invention, there are provided methods for the in vivo reduction of free radical levels in mammalian subjects in need thereof. In contrast to the inhibitory approach described in the prior art (i.e., wherein the function of the species responsible for free radical production is inhibited), the present invention employs a scavenging approach whereby overproduced free radical is bound in vivo to a suitable free radical scavenger. An exemplary free radical scavenger contemplated for use in the practice of the present invention is a dithiocarbamate-ferrous iron complex. This complex binds to free radicals, forming a stable, water-soluble free radical-containing complex. When administered to a subject afflicted with a disorder associated with free radical overproduction, the water-soluble free radical-containing complex is produced and then filtered through the kidneys, concentrated in the urine, and eventually excreted by the subject, thereby reducing in vivo free radical levels.

L113 ANSWER 13 OF 44 USPATFULL
ACCESSION NUMBER: 2002:137026 USPATFULL
TITLE: **Spin trapping** pharmaceutical compositions and methods for use thereof
INVENTOR(S): Carney, John M., Lexington, KY, United States
Floyd, Robert A., Oklahoma City, OK, United States
PATENT ASSIGNEE(S): Oklahoma Medical Research Foundation, Oklahoma City, OK, United States (U.S. corporation)
University of Kentucky Foundation, Lexington, KY, United States (U.S. corporation)

| | NUMBER | KIND | DATE |
|---|---|---|---|
| PATENT INFORMATION: | US 6403627 | B1 | 20020611 |
| APPLICATION INFO.: | US 1999-357297 | | 19990720 (9) |

RELATED APPLN. INFO.: Continuation of Ser. No. US 1997-969344, filed on 28 Nov 1997, now patented, Pat. No. US 6002001 Continuation of Ser. No. US 1994-167900, filed on 29 Jul 1994, now abandoned Continuation-in-part of Ser. No. US 1994-212800, filed on 15 Mar 1994, now patented,

Pat. No. US 5622994 Continuation of Ser. No. US 1993-52870, filed on 23 Apr 1993, now abandoned Continuation of Ser. No. US 1991-716952, filed on 18 Jun 1991, now abandoned
DOCUMENT TYPE: Utility
FILE SEGMENT: GRANTED
PRIMARY EXAMINER: Jones, Dwayne C.
LEGAL REPRESENTATIVE: Birch, Stewart, Kolasch & Birch, LLP
NUMBER OF CLAIMS: 20
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 0 Drawing Figure(s); 0 Drawing Page(s)
LINE COUNT: 920
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB **Spin trapping** compositions in general have now been discovered to be effective in treating a variety of disorders, including disorders such as those arising from ischemia, infection, inflammation, exposure to radiation or cytotoxic compounds, not just of the central and peripheral nervous systems but of peripheral organ disease having a wide variety of etiologies. In the preferred embodiment, the compositions for treating tissue damage from ischemia contain PBN, or active derivatives thereof, in a suitable pharmaceutical carrier for intravenous, oral, **topical**, or nasal/pulmonary administration. Other preferred **spin-trapping** agents include 5,5-dimethyl pyrroline N-oxide (DMPO), .alpha.-(4-pyridyl-1-oxide)-N-tert-butylnitrone (POBN), and (TEMPO) and **spin-trapping** derivatives thereof. Examples of derivatives of PBN include halogenated derivatives, bifunctional derivatives, conjugates with drugs or targeting molecules, dimers and cyclodextran polymers of PBN. Many different disorders can be treated using these compounds, including diseases or disorders of the central and peripheral nervous systems, and disorders arising from ischemia, infection, inflammation, oxidation from exposure to radiation or cytotoxic compounds, as well as due to naturally occurring processes such as aging.

IT **3376-24-7 3376-24-7D**, derivs.
(for nonsteroidal-antiinflammatory-caused gastric ulcer treatment)

L113 ANSWER 14 OF 44 USPATFULL
ACCESSION NUMBER: 2000:22943 USPATFULL
TITLE: **Spin trap** nitronyl hindered phenols
INVENTOR(S): Janzen, Edward G., Guelph, Canada
Wilcox, Allan L., Mountain View, CA, United States
PATENT ASSIGNEE(S): Oklahoma Medical Research Foundation, Oklahoma City, OK, United States (U.S. corporation)

| | NUMBER | KIND | DATE | |
|---|---|---|---|---|
| PATENT INFORMATION: | US 36594 | | 20000229 | |
| | US 5455272 | | 19951003 | (Original) |
| APPLICATION INFO.: | US 1997-942494 | | 19971002 | (8) |
| | US 1993-141241 | | 19931022 | (Original) |

DOCUMENT TYPE: Reissue
FILE SEGMENT: Granted
PRIMARY EXAMINER: O'Sullivan, Peter
NUMBER OF CLAIMS: 21
EXEMPLARY CLAIM: 1,3
NUMBER OF DRAWINGS: 3 Drawing Figure(s); 3 Drawing Page(s)
LINE COUNT: 628
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The invention is the use of nitronyl substituted hindered phenols as antioxidants in therapeutic applications. In the preferred embodiment the compositions have the general formula: ##STR1## Wherein R1 is hydrogen, an alkyl or an aryl and R2 is an alkyl or an aryl; R.sub.3 is an alkyl; and R.sub.4 is an alkyl. Further, the invention relates to

novel compositions useful as antioxidants. The novel compounds include: 2,6-di-tert-butyl-4-(N-tert-octyl)nitronyl phenol (DBONP); 2,6-dimethyl-4-(N-tert-octyl)nitronyl phenol (DMONP); N-tert-octyl-C-phenyl nitrone (OPN).

IT **3376-24-7**
(**spin trap** nitronyl-hindered phenols for therapeutic antioxidants)

L113 ANSWER 15 OF 44 USPATFULL
ACCESSION NUMBER: 1999:163856 USPATFULL
TITLE: **Spin trapping** pharmaceutical compositions and methods for use thereof
INVENTOR(S): Carney, John M., Saratoga, CA, United States
Floyd, Robert A., Oklahoma City, OK, United States
PATENT ASSIGNEE(S): Oklahoma Medical Research Foundation, Oklahoma City, OK, United States (U.S. corporation)
University of Kentucky Research Foundation, Lexington, KY, United States (U.S. corporation)

| | NUMBER | KIND | DATE |
|---|---|---|---|
| PATENT INFORMATION: | US 6002001 | | 19991214 |
| APPLICATION INFO.: | US 1997-969344 | | 19971128 (8) |

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 1991-716952, filed on 18 Jun 1991, now abandoned And a continuation of Ser. No. US 1994-167900, filed on 29 Jul 1994, now abandoned which is a continuation-in-part of Ser. No. US 1994-212800, filed on 15 Mar 1994, now patented, Pat. No. US 5622994 which is a continuation of Ser. No. US 1993-52870, filed on 26 Apr 1993, now abandoned which is a continuation of Ser. No. US 716952
DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Jones, Dwayne C.
NUMBER OF CLAIMS: 18
EXEMPLARY CLAIM: 1
LINE COUNT: 882
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB **Spin trapping** compositions in general have now been discovered to be effective in treating a variety of disorders, including disorders such as those arising from ischemia, infection, inflammation, exposure to radiation or cytotoxic compounds, not just of the central and peripheral nervous systems but of peripheral organ disease having a wide variety of etiologies. In the preferred embodiment, the compositions for treating tissue damage from ischemia contain PBN, or active derivatives thereof, in a suitable pharmaceutical carrier for intravenous, oral, **topical**, or nasal/pulmonary administration. Other preferred **spin-trapping** agents include 5,5-dimethyl pyrroline N-oxide, (DMPO), .alpha.-(4-pyridyl-1-oxide)-N-tert-butylnitrone, (POBN), and (TEMPO) **spin-trapping** derivatives thereof. Examples of derivatives of PBN include halogenated derivatives, bifunctional derivatives, conjugates with drugs or targeting molecules, dimers and cyclodextran polymers of PBN. Many different disorders can be treated using these compounds, including diseases or disorders of the central and peripheral nervous systems, and disorders arising from ischemia, infection, inflammation, oxidation from exposure to radiation or cytotoxic compounds, as well as due to naturally occurring processes such as aging.

IT **3376-24-7 3376-24-7D**, derivs.
(for nonsteroidal-antiinflammatory-caused gastric ulcer treatment)

L113 ANSWER 16 OF 44 USPATFULL
ACCESSION NUMBER: 1999:53439 USPATFULL

TITLE: Multicyclic nitrone **spin trapping** compositions
INVENTOR(S): Janzen, Edward G., Oklahoma City, OK, United States
Sankuratri, Nagaraju, Oklahoma City, OK, United States
PATENT ASSIGNEE(S): Oklahoma Medical Research Foundation, Oklahoma City, OK, United States (U.S. corporation)

| | NUMBER | KIND | DATE |
|---|---|---|---|
| PATENT INFORMATION: | US 5900227 | | 19990504 |
| APPLICATION INFO.: | US 1996-664450 | | 19960617 (8) |

DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Kight, John
ASSISTANT EXAMINER: Jones, Dameron
LEGAL REPRESENTATIVE: Arnall Golden & Gregory, LLP
NUMBER OF CLAIMS: 22
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 4 Drawing Figure(s); 4 Drawing Page(s)
LINE COUNT: 1103
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB Multicyclic nitrone **spin trapping** compounds capable of forming stable free radical spin adducts are provided as well as methods for their synthesis. The multicyclic nitrone **spin trapping** compounds can be reacted with a free radical, such as a hydroxy or hydroperoxy radical, in solution to form a spin adduct which is stable and readily detectable by electron paramagnetic resonance (EPR) spectroscopy. The multicyclic nitrone **spin traps** can be used to detect free radicals in a sample such as a biological system. In one embodiment, the **spin trapping** compound, 1,3,3-trimethyl-6-azabicyclo-[3.2.1]oct-6-ene-N-oxide ("TRAZON") is provided, as well as methods for the synthesis of TRAZON and of modified forms of TRAZON. TRAZON can react with a wide range of different free radicals in solution to form free radical spin adducts which are readily detectable by EPR.

L113 ANSWER 17 OF 44 USPATFULL
ACCESSION NUMBER: 1998:22262 USPATFULL
TITLE: **Topical spin trap** composition and method
INVENTOR(S): Proctor, Peter H., 4126 Southwest Freeway, Ste. 1616, Houston, TX, United States 77027

| | NUMBER | KIND | DATE |
|---|---|---|---|
| PATENT INFORMATION: | US 5723502 | | 19980303 |
| APPLICATION INFO.: | US 1995-465411 | | 19950605 (8) |

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 1994-229374, filed on 18 Apr 1994, now patented, Pat. No. US 5470876 And Ser. No. US 1994-193228, filed on 7 Feb 1994, now patented, Pat. No. US 5472687 , each Ser. No. US - which is a continuation-in-part of Ser. No. US 1993-21970, filed on 24 Feb 1993, now patented, Pat. No. US 5352442 which is a continuation-in-part of Ser. No. US 1988-149720, filed on 29 Jan 1988, now abandoned which is a continuation-in-part of Ser. No. US 1987-8186, filed on 28 Jan 1987, now abandoned which is a continuation-in-part of Ser. No. US 1986-858050, filed on 30 Apr 1986, now abandoned which is a continuation-in-part of Ser. No. US 1985-757131, filed on 18 Jul 1985, now abandoned
DOCUMENT TYPE: Utility
FILE SEGMENT: Granted

PRIMARY EXAMINER: Lambkin, Deborah
LEGAL REPRESENTATIVE: Lundeen, Daniel N.Sroufe, Payne & Lundeen, L.L.P.
NUMBER OF CLAIMS: 17
EXEMPLARY CLAIM: 1
LINE COUNT: 332
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB A composition and method for ameliorating a cellular dysfunction of a tissue such as the cosmetic treatment of hair loss and stimulation of hair growth are disclosed. The method comprises administering a nitroso or nitrone **spin trap** such as N-t-butyl-.alpha.-phenylnitrone (PBN) to the affected tissue.
IT **3317-61-1**, 5,5-Dimethyl-1-pyrroline N-oxide **3376-24-7**, N-tert-Butyl-.alpha.-phenylnitrone **66893-81-0**
(**topical spin trap** compn. for treatment of hair loss and stimulation of hair growth)

L113 ANSWER 18 OF 44 USPATFULL
ACCESSION NUMBER: 97:96884 USPATFULL
TITLE: Use of a **spin trap** in a cosmetic or dermatological composition
INVENTOR(S): Ribier, Alain, Paris, France
Nguyen, Quang Lan, Antony, France
Simonnet, Jean-Thierry, Paris, France
Boussouira, Boudiaf, Paris, France
PATENT ASSIGNEE(S): L'Oreal, Paris, France (non-U.S. corporation)

| | NUMBER | KIND | DATE |
|---|---|---|---|
| PATENT INFORMATION: | US 5679691 | | 19971021 |
| APPLICATION INFO.: | US 1996-597101 | | 19960206 (8) |

RELATED APPLN. INFO.: Division of Ser. No. US 1994-366748, filed on 30 Dec 1994, now patented, Pat. No. US 5569663

| | NUMBER | DATE |
|---|---|---|
| PRIORITY INFORMATION: | FR 1993-15869 | 19931230 |

DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Dodson, Shelley A.
LEGAL REPRESENTATIVE: Oblon, Spivak, McClelland, Maier & Neustadt, P.C.
NUMBER OF CLAIMS: 18
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 1 Drawing Figure(s); 1 Drawing Page(s)
LINE COUNT: 624
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The invention relates to the use of a **spin trap** employed as an electron paramagnetic resonance measurement probe, in a cosmetic or dermatological composition for the light-protective, anti-ageing and/or anti-acne treatment of the **skin**. In particular, this **spin trap** is encapsulated in lipid vesicles which are capable of diffusing into the deep layers of the **skin**.
IT **2564-83-2**
(cosmetic and pharmaceutical compns. contg. spin probes)

L113 ANSWER 19 OF 44 USPATFULL
ACCESSION NUMBER: 97:33789 USPATFULL
TITLE: **Spin trapping** pharmaceutical compositions and methods for use thereof
INVENTOR(S): Carney, John M., Lexington, KY, United States
Floyd, Robert A., Oklahoma City, OK, United States
PATENT ASSIGNEE(S): Oklahoma Medical Research Foundation, Oklahoma City, OK, United States (U.S. corporation)

University of Kentucky Research Foundation, Lexington, KY, United States (U.S. corporation)

| | NUMBER | KIND | DATE |
|---|---|---|---|
| PATENT INFORMATION: | US 5622994 | | 19970422 |
| APPLICATION INFO.: | US 1994-212800 | | 19940315 (8) |

RELATED APPLN. INFO.: Continuation of Ser. No. US 1993-52870, filed on 26 Apr 1993, now abandoned which is a continuation of Ser. No. US 1991-716952, filed on 18 Jun 1991, now abandoned which is a continuation-in-part of Ser. No. US 1990-589177, filed on 27 Sep 1990, now abandoned which is a continuation-in-part of Ser. No. US 1989-422651, filed on 17 Oct 1989, now patented, Pat. No. US 5025032
DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Burn, Brian M.
NUMBER OF CLAIMS: 36
EXEMPLARY CLAIM: 1
LINE COUNT: 880
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB **Spin trapping** compositions in general have now been discovered to be effective in treating a variety of disorders, including disorders such as those arising from ischemia, infection, inflammation, exposure to radiation or cytotoxic compounds, not just of the central and peripheral nervous systems but of peripheral organ disease having a wide variety of etiologies. In the preferred embodiment, the compositions for treating tissue damage from ischemia contain PBN, or active derivatives thereof, in a suitable pharmaceutical carrier for intravenous, oral, **topical**, or nasal/pulmonary administration. Many different disorders can be treated using these compounds, including diseases or disorders of the central and peripheral nervous systems, and disorders arising from ischemia, infection, inflammation, oxidation from exposure to radiation or cytotoxic compounds, as well as due to naturally occurring processes such as aging.

IT **3376-24-7 3376-24-7D**, derivs.
(for nonsteroidal-antiinflammatory-caused gastric ulcer treatment)

L113 ANSWER 20 OF 44 USPATFULL
ACCESSION NUMBER: 96:99215 USPATFULL
TITLE: Use of a **spin trap** in a cosmetic or dermatological composition
INVENTOR(S): Ribier, Alain, Paris, France
Nguyen, Quang L., Antony, France
Simonnet, Jean-Thierry, Paris, France
Boussouira, Boudiaf, Paris, France
PATENT ASSIGNEE(S): L'Oreal, Paris, France (non-U.S. corporation)

| | NUMBER | KIND | DATE |
|---|---|---|---|
| PATENT INFORMATION: | US 5569663 | | 19961029 |
| APPLICATION INFO.: | US 1994-366748 | | 19941230 (8) |

| | NUMBER | DATE |
|---|---|---|
| PRIORITY INFORMATION: | FR 1993-15869 | 19931230 |

DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Dodson, Shelley A.
LEGAL REPRESENTATIVE: Oblon, Spivak, McClelland, Maier & Neustadt, P.C.
NUMBER OF CLAIMS: 13
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 1 Drawing Figure(s); 1 Drawing Page(s)

LINE COUNT: 620
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The invention relates to the use of a **spin trap** employed as an electron paramagnetic resonance measurement probe, in a cosmetic or dermatological composition for the light-protective, anti-ageing and/or anti-acne treatment of the **skin**. In particular, this **spin trap** is encapsulated in lipid vesicles which are capable of diffusing into the deep layers of the **skin**.
IT **2564-83-2**
(cosmetic and pharmaceutical compns. contg. spin probes)

L113 ANSWER 21 OF 44 USPATFULL
ACCESSION NUMBER: 91:40444 USPATFULL
TITLE: Method for production of graft copolymer, pattern replication method, and base polymer and resist for graft copolymerization
INVENTOR(S): Soda, Yasunari, Hachioji, Japan
Mochiji, Kozo, Hachioji, Japan
Oizumi, Hiroaki, Kokubunji, Japan
Kimura, Takeshi, Higashimurayama, Japan
PATENT ASSIGNEE(S): Hitachi, Ltd., Tokyo, Japan (non-U.S. corporation)

| | NUMBER | KIND | DATE |
|---|---|---|---|
| PATENT INFORMATION: | US 5017458 | | 19910521 |
| APPLICATION INFO.: | US 1989-354116 | | 19890522 (7) |

| | NUMBER | DATE |
|---|---|---|
| PRIORITY INFORMATION: | JP 1988-128394 | 19880527 |

DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Hamilton, Cynthia
LEGAL REPRESENTATIVE: Antonelli, Terry, Stout & Kraus
NUMBER OF CLAIMS: 9
EXEMPLARY CLAIM: 1,9
NUMBER OF DRAWINGS: 7 Drawing Figure(s); 2 Drawing Page(s)
LINE COUNT: 273
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The method for production of a graft copolymer according to the present invention includes the step of adding to a base polymer capable of forming first radicals when irradiated with radiation an additive capable of combining with said first radicals to form second radicals stable against oxygen, the step of irradiating said base polymer containing the additive with radiation, and the step of introducing a monomer under an atmosphere free from oxygen, thereby to graft copolymerize said irradiated base polymer and said monomer.
IT **3317-61-1**, 5,5-Dimethyl-1-pyrroline-1-oxide
(**spin traps**, for radiochem. grafting in presence of oxygen)

L113 ANSWER 22 OF 44 MEDLINE DUPLICATE 2
ACCESSION NUMBER: 1998025413 MEDLINE
DOCUMENT NUMBER: 98025413 PubMed ID: 9378364
TITLE: Oxidative damage to nucleic acids photosensitized by titanium dioxide.
AUTHOR: Wamer W G; Yin J J; Wei R R
CORPORATE SOURCE: Center for Food Safety and Applied Nutrition, U.S. Food and Drug Administration, Washington, DC 20204, USA..

WGW@FDACF.SSW.DHHS.GOV
SOURCE: FREE RADICAL BIOLOGY AND MEDICINE, (1997) 23 (6) 851-8.
Journal code: 8709159. ISSN: 0891-5849.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199711
ENTRY DATE: Entered STN: 19971224
Last Updated on STN: 19971224
Entered Medline: 19971112

ABSTRACT:
The semiconductor TiO2 is known to have photobiological activity in prokaryotic and eukaryotic cells. Applications of this photobiological activity have been suggested including sterilization of waste water and phototherapy of malignant cells. Here, several model and cellular systems were used to study the mechanism of photocatalysis by TiO2. Treatment of TiO2 (anatase, 0.45 microns), suspended in water containing a **spin trap** 5,5-dimethyl-1-pyrroline-N-oxide (DMPO), with UV radiation (320 nm) resulted in an electron spin resonance (ESR) signal characteristic of the hydroxyl radical. Irradiation of solutions containing calf thymus DNA and TiO2 with UVA (320-400 nm) radiation resulted in hydroxylation of guanine bases. The degree of hydroxylation was dependent on both UVA fluence and amount of TiO2 in suspension. Human skin fibroblasts, preincubated 18 h with 10 micrograms/cm2 TiO2 and then UVA-irradiated (0-58 KJ/m2), showed dose dependent photocytoxicity. RNA, isolated from similarly treated fibroblasts, contained significant levels of photooxidation, measured as hydroxylation of guanine bases. However, no oxidative damage was detectable in cellular DNA. These results suggest that nucleic acids are a potential target for photooxidative damage sensitized by TiO2, and support the view that TiO2 photocatalyzes free radical formation.

CONTROLLED TERM: Check Tags: Animal; Human
Cattle
Cell Line
Cyclic N-Oxides
DNA: DE, drug effects
DNA: ME, metabolism
DNA: RE, radiation effects
Deoxyguanine Nucleotides: ME, metabolism
Deoxyguanine Nucleotides: RE, radiation effects
Dose-Response Relationship, Drug
Dose-Response Relationship, Radiation
Electron Spin Resonance Spectroscopy
Fibroblasts: DE, drug effects
Fibroblasts: RE, radiation effects
*Nucleic Acids: DE, drug effects
Nucleic Acids: RE, radiation effects
***Oxidative Stress: DE, drug effects**
**Oxidative Stress: RE, radiation effects**
Photochemistry
Photosensitizing Agents: RE, radiation effects
*Photosensitizing Agents: TO, toxicity
RNA: DE, drug effects
RNA: ME, metabolism
RNA: RE, radiation effects
**Skin**
Spin Labels
Suspensions
Titanium: RE, radiation effects
*Titanium: TO, toxicity
Ultraviolet Rays
CAS REGISTRY NO.: 13463-67-7 (titanium dioxide); 139307-94-1

(8-oxodeoxyguanosine triphosphate); **3317-61-1 (5,5-dimethyl-1-pyrroline-1-oxide)**; 63231-63-0 (RNA); 7440-32-6 (Titanium); 9007-49-2 (DNA)

CHEMICAL NAME: 0 (Cyclic N-Oxides); 0 (Deoxyguanine Nucleotides); 0 (Nucleic Acids); 0 (Photosensitizing Agents); 0 (Spin Labels); 0 (Suspensions)

L113 ANSWER 23 OF 44 MEDLINE DUPLICATE 3

ACCESSION NUMBER: 97059355 MEDLINE

DOCUMENT NUMBER: 97059355 PubMed ID: 8903676

TITLE: Free radical reactions photosensitized by the human lens component, kynurenine: an EPR and **spin trapping** investigation.

AUTHOR: Reszka K J; Bilski P; Chignell C F; Dillon J

CORPORATE SOURCE: Laboratory of Molecular Biophysics, National Institute of Environmental Health Sciences, National Institute of Health, Research Triangle Park, NC, USA. REXZKA@NIEHS.NIH.GOV. RESZKA@NIEHS.NIH.GOV

SOURCE: FREE RADICAL BIOLOGY AND MEDICINE, (1996) 20 (1) 23-34. Journal code: 8709159. ISSN: 0891-5849.

PUB. COUNTRY: United States

DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English

FILE SEGMENT: Priority Journals

ENTRY MONTH: 199703

ENTRY DATE: Entered STN: 19970327
Last Updated on STN: 20000303
Entered Medline: 19970314

ABSTRACT:
We have undertaken electron paramagnetic resonance and **spin** ***trapping*** investigations of the photochemistry of kynurenine (KN), a natural component of the human eye and close analog of the principal chromophore in the young human lens 3-OH-kynurenine O-glucoside (3HKG). 5,5-Dimethyl-1-pyrroline N-oxide (DMPO) was employed as a **spin** ***trap*** . We found that upon UV irradiation (> 300 nm) KN photoreduces oxygen to superoxide radical (in DMSO) and nitromethane ($CH_3NO_2$) to a nitromethane radical anion ($CH_3NO_2^{\cdot-}$) (in air-free buffers, pH 7 and 9.5). KN also sensitized photooxidation of cysteine, NADH, EDTA, azide, and ascorbate; oxygen greatly accelerated this process. Oxidation of cysteine, NADH, and EDTA was accompanied by superoxide radical formation. Cysteinyl and azidyl radicals were detected as DMPO adducts. We also observed that KN undergoes photodegradation to a product(s) whose photosensitizing capacity is greater than that of KN itself. We postulate that: (i) 3HKG may be able to photoinitiate free radical reactions in vivo, and (ii) oxygen is an important factor determining the yields of free radical processes initiated by lenticular chromophores.

CONTROLLED TERM: Check Tags: Human
Ascorbic Acid: ME, metabolism
Cyclic N-Oxides: ME, metabolism
Cysteine: ME, metabolism
*Electron Spin Resonance Spectroscopy
Electron Transport
Eye: ME, metabolism
Free Radicals: ME, metabolism
*Kynurenine: PD, pharmacology
Lens, Crystalline: CH, chemistry
Methane: AA, analogs & derivatives
Methane: ME, metabolism
Models, Chemical
Molecular Structure
Nitroparaffins: ME, metabolism
Oxidation-Reduction

Oxygen: AN, analysis
Oxygen: ME, metabolism
Photochemistry
***Photosensitivity Disorders: ME, metabolism**
Singlet Oxygen
Spectrophotometry
Spin Labels
Superoxide Dismutase: ME, metabolism
Superoxides: ME, metabolism
Ultraviolet Rays

CAS REGISTRY NO.: 11062-77-4 (Superoxides); 17778-80-2 (Singlet Oxygen); **3317-61-1 (5,5-dimethyl-1-pyrroline-1-oxide);** 343-65-7 (Kynurenine); 50-81-7 (Ascorbic Acid); 52-90-4 (Cysteine); 74-82-8 (Methane); 75-52-5 (nitromethane); 7782-44-7 (Oxygen)

CHEMICAL NAME: 0 (Cyclic N-Oxides); 0 (Free Radicals); 0 (Nitroparaffins); 0 (Spin Labels); EC 1.15.1.1 (Superoxide Dismutase)

L113 ANSWER 24 OF 44 MEDLINE DUPLICATE 4
ACCESSION NUMBER: 95221937 MEDLINE
DOCUMENT NUMBER: 95221937 PubMed ID: 7706763
TITLE: Effect of topically applied tocopherol on ultraviolet radiation-mediated free radical damage in skin.
AUTHOR: Jurkiewicz B A; Bissett D L; Buettner G R
CORPORATE SOURCE: Radiation Research Laboratory, University of Iowa College of Medicine, Iowa City 52242-1101, USA.
SOURCE: JOURNAL OF INVESTIGATIVE DERMATOLOGY, (1995 Apr) 104 (4) 484-8.
Journal code: 0426720. ISSN: 0022-202X.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199505
ENTRY DATE: Entered STN: 19950518
Last Updated on STN: 19960129
Entered Medline: 19950509

ABSTRACT:
Previously, we demonstrated by electron paramagnetic resonance (EPR) spectroscopy that ultraviolet radiation induces free-radical formation in Skh-1 hairless mouse skin. Because free-radical oxidative stress is thought to play a principal role in skin photoaging and cancer, oxidative stress and subsequent photodamage should be decreased by supplementation of skin with antioxidants. Using both the ascorbate free radical and an EPR **spin-***trapping***** system to detect short-lived radicals, we evaluated the effect of the topically applied antioxidants tocopherol sorbate, alpha-tocopherol, and tocopherol acetate on ultraviolet radiation-induced free-radical formation. We show that tocopherol sorbate significantly decreases the ultraviolet radiation-induced radical flux in skin. With our chronically exposed mouse model, tocopherol sorbate was also found to be significantly more protective against skin photoaging than alpha-tocopherol and tocopherol acetate. These results extend our previous observations of ultraviolet radiation-induced free-radical generation in skin and indicate the utility of tocopherol sorbate as an antioxidant in providing significant protection against ultraviolet radiation-induced oxidative damage.

CONTROLLED TERM: Check Tags: Animal; Female; Support, Non-U.S. Gov't
**Administration, Topical**
Aging
Ascorbic Acid: ME, metabolism
Free Radicals
Mice
Mice, Inbred HRS

Oxidative Stress: DE, drug effects
*Skin: RE, radiation effects
*Ultraviolet Rays: AE, adverse effects
Vitamin E: AD, administration & dosage
*Vitamin E: PD, pharmacology
CAS REGISTRY NO.: 1406-18-4 (Vitamin E); 50-81-7 (Ascorbic Acid)
CHEMICAL NAME: 0 (Free Radicals)

L113 ANSWER 25 OF 44 MEDLINE
ACCESSION NUMBER: 2002324568 MEDLINE
DOCUMENT NUMBER: 22055451 PubMed ID: 12060808
TITLE: The **spin trapping** agent PBN stimulates H2 O2 -induced Erk and Src kinase activity in human neuroblastoma cells.
AUTHOR: Kelicen Pelin; Cantuti-Castelvetri Ippolita; Pekiner Can; Paulson K Eric
CORPORATE SOURCE: Karolinska Institutet, Division of Molecular Toxicology, Institute of Environmental Medicine, S-171 77 Stockholm, Sweden.
SOURCE: NEUROREPORT, (2002 Jun 12) 13 (8) 1057-61.
Journal code: 9100935. ISSN: 0959-4965.
PUB. COUNTRY: England: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200207
ENTRY DATE: Entered STN: 20020618
Last Updated on STN: 20020727
Entered Medline: 20020726

ABSTRACT:
The **spin-trap**, alpha-phenyl-N-tert-butylnitrone (PBN) has been shown to have neuroprotective properties and may prevent oxidative injury in vivo and in cultured cells. Although PBN quenches reactive oxygen species, the direct mechanism of neuroprotective action is unknown. In the present study, we examined the effects of PBN on the regulation of the mitogen activated kinase Erk and as well as Src family tyrosine kinases, enzymes known to be activated by oxygen species such as H2O2. In SH-SY5Y human neuroblastoma cells, H2O2 induced activation of Erk and Src kinases was markedly potentiated by treatment with PBN. The potentiation by PBN of the Erk and Src kinase activation by H2O2 required extracellular Ca2+ and appeared dependent on voltage sensitive Ca2+ channels. In contrast, PBN did not affect depolarization-dependent or growth factor-dependent Erk and Src kinase phosphorylation. Our results suggest that PBN might have a protective effect on cells by potentiating the anti-apoptotic Erk and Src kinase pathways responding to H2O2, an effect apparently distinct from its ability to trap oxygen free radicals.

CONTROLLED TERM: Check Tags: Human; Support, Non-U.S. Gov't
Calcium: ME, metabolism
Calcium: PD, pharmacology
Calcium Channels, L-Type: DE, drug effects
Calcium Channels, L-Type: ME, metabolism
Central Nervous System: DE, drug effects
Central Nervous System: EN, enzymology
Central Nervous System: PP, physiopathology
Drug Interactions: PH, physiology
Epidermal Growth Factor: PD, pharmacology
Extracellular Space: DE, drug effects
Extracellular Space: ME, metabolism
*Hydrogen Peroxide: PD, pharmacology
Membrane Potentials: DE, drug effects
Membrane Potentials: PH, physiology
*Mitogen-Activated Protein Kinases: DE, drug effects

Mitogen-Activated Protein Kinases: ME, metabolism
Neuroblastoma
*Neurodegenerative Diseases: DT, drug therapy
Neurodegenerative Diseases: EN, enzymology
Neurodegenerative Diseases: PP, physiopathology
Neurons: DE, drug effects
Neurons: EN, enzymology
*Neuroprotective Agents: PD, pharmacology
*Nitrogen Oxides: PD, pharmacology
***Oxidative Stress: DE, drug effects**
Oxidative Stress: PH, physiology
Potassium Chloride: PD, pharmacology
Tumor Cells, Cultured
*src-Family Kinases: DE, drug effects
src-Family Kinases: ME, metabolism

CAS REGISTRY NO.: **3376-24-7 (phenyl-N-tert-butylnitrone);** 62229-50-9 (Epidermal Growth Factor); 7440-70-2 (Calcium); 7447-40-7 (Potassium Chloride); 7722-84-1 (Hydrogen Peroxide)

CHEMICAL NAME: 0 (Calcium Channels, L-Type); 0 (Neuroprotective Agents); 0 (Nitrogen Oxides); EC 2.7.1.- (Mitogen-Activated Protein Kinases); EC 2.7.11.- (src-Family Kinases)

L113 ANSWER 26 OF 44 MEDLINE
ACCESSION NUMBER: 2001692895 MEDLINE
DOCUMENT NUMBER: 21569882 PubMed ID: 11712911
TITLE: Photochemistry and photocytotoxicity of alkaloids from Goldenseal (Hydrastis canadensis L.) 1. Berberine.
AUTHOR: Inbaraj J J; Kukielczak B M; Bilski P; Sandvik S L; Chignell C F
CORPORATE SOURCE: Laboratory of Pharmacology and Chemistry, National Institute of Environmental Health Sciences, National Institutes of Health, Research Triangle Park, NC 27709, USA.
SOURCE: CHEMICAL RESEARCH IN TOXICOLOGY, (2001 Nov) 14 (11) 1529-34.
Journal code: 8807448. ISSN: 0893-228X.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200112
ENTRY DATE: Entered STN: 20011217
Last Updated on STN: 20020403
Entered Medline: 20011226

ABSTRACT:
Goldenseal is an herb which is widely used for many medical applications such as in eyewashes and skin lotions and which is currently undergoing testing by the National Toxicology Program. The main alkaloid constituent of Goldenseal is berberine. The topical application of Goldenseal or berberine to the skin or eyes raises the possibility that an adverse phototoxic reaction may result from an interaction between the alkaloid and light. We have therefore studied the photochemistry of berberine in different solvents and its phototoxicty to HaCaT keratinocytes. Irradiation of berberine in aqueous solutions does not generate (1)O(2), but in CH(2)Cl(2), (1)O(2) is produced with a quantum yield varphi = 0.34. With the aid of the electron paramagnetic resonance (EPR) **spin** ***trapping*** technique and 5,5-dimethyl-1-pyrroline N-oxide (DMPO), we have detected oxygen-centered radicals photogenerated by berberine in water and acetonitrile. In the latter solvent and in the absence of oxygen, the neutral berberine radical formed by one electron reduction was observed. Methanol radicals were detected by EPR in water/alcohol low-temperature glasses irradiated in the berberine long-wavelength absorption band. In such alcoholic glasses, we have also detected an EPR signal from the berberine triplet at 77

K, in contrast to aqueous glasses where neither triplet nor radicals were detectable. Our data show that, although a weak photosensitizer in water, berberine is able to produce both (1)O(2) and radical species in a nonpolar environment. UVA irradiation of HaCaT keratinocytes in the presence of 50 microM berberine resulted in an 80% decrease in cell viability and a 3-fold increase in DNA damage as measured by the Comet assay. These findings suggest that exposure to sunlight or artificial light sources emitting UVA should be avoided when topical preparations derived from Goldenseal or containing berberine are used.

CONTROLLED TERM: Check Tags: Human
**Administration, Topical**
*Berberine: CH, chemistry
*Berberine: TO, toxicity
Cell Culture
Cell Survival
*DNA Damage
*Dermatitis, Phototoxic: PP, physiopathology
Electron Spin Resonance Spectroscopy
Free Radicals
Keratinocytes: PA, pathology
Oxidation-Reduction
Photochemistry
Photosensitizing Agents: CH, chemistry
Plant Extracts: CH, chemistry
Plant Extracts: TO, toxicity
*Ranunculaceae: CH, chemistry
Solvents
Ultraviolet Rays
CAS REGISTRY NO.: 2086-83-1 (Berberine)
CHEMICAL NAME: 0 (Free Radicals); 0 (Photosensitizing Agents); 0 (Plant Extracts); 0 (Solvents)

L113 ANSWER 27 OF 44 MEDLINE
ACCESSION NUMBER: 2001176805 MEDLINE
DOCUMENT NUMBER: 21028160 PubMed ID: 11156442
TITLE: Preventive effect of several antioxidants after oxidative stress on rat brain homogenates.
AUTHOR: Horakova L; Ondrejickova O; Bachrata K; Vajdova M
CORPORATE SOURCE: Institute of Experimental Pharmacology, Slovak Academy of Sciences, Bratislava.. exfahorl@savba.sk
SOURCE: GENERAL PHYSIOLOGY AND BIOPHYSICS, (2000 Jun) 19 (2) 195-205.
Journal code: 8400604. ISSN: 0231-5882.
PUB. COUNTRY: Slovakia
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200103
ENTRY DATE: Entered STN: 20010404
Last Updated on STN: 20010404
Entered Medline: 20010329

ABSTRACT:
Brain homogenate was used as a model system to study antioxidant properties of several natural and synthetic antioxidants under oxidative stress. Oxidative stress was induced by Fe/ascorbate system and lipid peroxidation as well as protein modification were studied. Thiobarbituric acid reactive substances (TBARS) were used as a marker of lipid peroxidation. The preventive effect concerning lipid peroxidation decreased in the order: buthylated hydroxytoluene (BHT) (3.5), stobadine (ST) (35), serotonin (54), trolox (98), U 74389G (160), melatonin (3100), (the numbers in the brackets represent IC50 in micromol/l). Methylprednisolone had no effect, and **spin traps** interfered with TBARS determination. Concerning creatine kinase (CK) activity as a

selected marker of oxidative modification of proteins, the preventive effect of antioxidants (30 micromol/l) decreased in the order: BHT (30), trolox (75), stobadine (ST) (77), alpha-phenyl-N-tert-buthylnitrone (PBN) (87), sodium salt of N-tert-buthyl-C-(phenyl-2-sulfone) nitrone (SPBN) (90), (the numbers in the brackets represent the loss of CK activity in percentages, when 100% was the loss of CK activity in the absence of any antioxidant). The nonglucocorticoid steroid U 74389G, methylprednisolone and serotonin had no preventive effects, while melatonin had antioxidant effect only in a higher concentration (1 mmol/l).

CONTROLLED TERM: Check Tags: Animal; Male; Support, Non-U.S. Gov't
*Antioxidants: PD, pharmacology
Benzenesulfonates: PD, pharmacology
*Brain: DE, drug effects
*Brain Injuries: PC, prevention & control
Butylated Hydroxytoluene: PD, pharmacology
Carbolines: PD, pharmacology
Chromans: PD, pharmacology
Creatine Kinase: ME, metabolism
Inhibitory Concentration 50
Lipid Peroxidation: DE, drug effects
Melatonin: PD, pharmacology
Methylprednisolone: PD, pharmacology
Models, Chemical
Neuroprotective Agents: PD, pharmacology
Nitrogen Oxides: PD, pharmacology
***Oxidative Stress: DE, drug effects**
Oxygen: ME, metabolism
Pregnatrienes: PD, pharmacology
Rats
Rats, Wistar
Serotonin: PD, pharmacology
Thiobarbituric Acid Reactive Substances: ME, metabolism

CAS REGISTRY NO.: 111668-89-4 (U 74389F); 113443-50-8 (N-tert-butyl-(2-sulfophenyl)nitrone); 128-37-0 (Butylated Hydroxytoluene); 17411-19-7 (dicarbine); **3376-24-7 (phenyl-N-tert-butylnitrone)**; 50-67-9 (Serotonin); 56305-04-5 (6-hydroxy-2,5,7,8-tetramethylchroman-2-carboxylic acid); 73-31-4 (Melatonin); 7782-44-7 (Oxygen); 83-43-2 (Methylprednisolone)

CHEMICAL NAME: 0 (Antioxidants); 0 (Benzenesulfonates); 0 (Carbolines); 0 (Chromans); 0 (Neuroprotective Agents); 0 (Nitrogen Oxides); 0 (Pregnatrienes); 0 (Thiobarbituric Acid Reactive Substances); EC 2.7.3.2 (Creatine Kinase)

L113 ANSWER 28 OF 44 MEDLINE
ACCESSION NUMBER: 1999412861 MEDLINE
DOCUMENT NUMBER: 99412861 PubMed ID: 10483363
TITLE: Ruby laser irradiation (694 nm) of human skin biopsies: assessment by electron spin resonance spectroscopy of free radical production and oxidative stress during laser depilation.
AUTHOR: Haywood R M; Wardman P; Gault D T; Linge C
CORPORATE SOURCE: RAFT Institute of Plastic Surgery, Mount Vernon Hospital, Northwood, Middlesex, UK.. heywood@raft.ac.uk
SOURCE: PHOTOCHEMISTRY AND PHOTOBIOLOGY, (1999 Sep) 70 (3) 348-52. Journal code: 0376425. ISSN: 0031-8655.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199910
ENTRY DATE: Entered STN: 19991014

Last Updated on STN: 19991014
Entered Medline: 19991007

ABSTRACT:
Human skin biopsies (hair-bearing scalp skin and non-hair-bearing breast skin) were treated with t-butylhydroperoxide, irradiated with UV light (UVR) or irradiated with 694 nm ruby laser red light. Free-radical production and oxidative stress were assessed with electron spin resonance spectroscopy (ESR) using the ascorbate radical as a marker. In comparison with both UVR and t-butyl-hydroperoxide (which readily induce the ascorbate radical in hair-bearing and hairless skin), 694 nm red light does not result in the formation of the ascorbate radical in detectable concentrations. **Spin -trapping** experiments with the **spin trap** 5,5-dimethyl-1-pyrroline N-oxide (DMPO) showed that while free radicals could be detected after treatment of skin with t-butylhydroperoxide, radicals could not be trapped after laser treatment. Treatment of lasered skin (containing DMPO) with t-butylhydroperoxide produced radical adducts as well as the ascorbate radical, demonstrating that the laser neither depletes endogenous ascorbate nor the preadministered **spin trap**. It is concluded that 694 nm red light does not induce oxidative stress in human skin in levels comparable either to t-butyl hydroperoxide or UV light.

CONTROLLED TERM: Check Tags: Comparative Study; Human; Support, Non-U.S. Gov't
Cyclic N-Oxides
Electron Spin Resonance Spectroscopy
Free Radicals: ME, metabolism
*Hair Removal: AE, adverse effects
*Lasers: AE, adverse effects
***Oxidative Stress**
***Skin: ME, metabolism**
***Skin: RE, radiation effects**
Spin Labels
**Spin Trapping**
Ultraviolet Rays
tert-Butylhydroperoxide: ME, metabolism
tert-Butylhydroperoxide: TO, toxicity

CAS REGISTRY NO.: **3317-61-1 (5,5-dimethyl-1-pyrroline-1-oxide);** 75-91-2 (tert-Butylhydroperoxide)

CHEMICAL NAME: 0 (Cyclic N-Oxides); 0 (Free Radicals); 0 (Spin Labels)

L113 ANSWER 29 OF 44 MEDLINE

ACCESSION NUMBER: 1998023083 MEDLINE

DOCUMENT NUMBER: 98023083 PubMed ID: 9358246

TITLE: alpha-Phenyl-N-tert-butylnitrone attenuates excitotoxicity in rat striatum by preventing hydroxyl radical accumulation.

AUTHOR: Lancelot E; Revaud M L; Boulu R G; Plotkine M; Callebert J

CORPORATE SOURCE: Laboratoire de Pharmacologie, Universite Descartes, Paris France.

SOURCE: FREE RADICAL BIOLOGY AND MEDICINE, (1997) 23 (7) 1031-4.
Journal code: 8709159. ISSN: 0891-5849.

PUB. COUNTRY: United States

DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English

FILE SEGMENT: Priority Journals

ENTRY MONTH: 199712

ENTRY DATE: Entered STN: 19980109
Last Updated on STN: 19980109
Entered Medline: 19971218

ABSTRACT:
Various in vitro experiments have indicated that oxygen-derived free radicals may contribute to excitotoxic neuronal death. In the present study we induced excitotoxicity in rat striatum by perfusing glutamate at a high concentration

through a microdialysis probe. We observed an increased formation of hydroxyl radicals (.OH) during the perfusion of the excitotoxin and an extensive striatal lesion 24 h after the insult. The **spin trap**, alpha-phenyl-N-tert-butylnitrone (PBN), attenuated both hydroxyl radical levels and the volume of the lesion. This result suggests that the neuroprotection may be due to a free radical scavenging mechanism. It also implies that PBN may be used in pathological situations involving excitotoxicity such as stroke, brain trauma, and chronic neurologic diseases.

CONTROLLED TERM: Check Tags: Animal; Comparative Study; Male
*Corpus Striatum: DE, drug effects
*Excitatory Amino Acids: TO, toxicity
Free Radical Scavengers
Hydroxyl Radical
Microdialysis
*Neuroprotective Agents: PD, pharmacology
*Nitrogen Oxides: PD, pharmacology
***Oxidative Stress: DE, drug effects**
Perfusion
Rats
Rats, Sprague-Dawley
Spin Labels

CAS REGISTRY NO.: 3352-57-6 (Hydroxyl Radical); **3376-24-7 (phenyl-N-tert-butylnitrone)**

CHEMICAL NAME: 0 (Excitatory Amino Acids); 0 (Free Radical Scavengers); 0 (Neuroprotective Agents); 0 (Nitrogen Oxides); 0 (Spin Labels)

L113 ANSWER 30 OF 44 MEDLINE

ACCESSION NUMBER: 97058192 MEDLINE

DOCUMENT NUMBER: 97058192 PubMed ID: 8902521

TITLE: The effects of alpha-phenyl-tert-butyl nitrone (PBN) on copper-induced rat fulminant hepatitis with jaundice.

AUTHOR: Yamashita T; Ohshima H; Asanuma T; Inukai N; Miyoshi I; Kasai N; Kon Y; Watanabe T; Sato F; Kuwabara M

CORPORATE SOURCE: Department of Animal Disease Control, Laboratory of Radiation Biology, Graduate School of Veterinary Medicine, Hokkaido University, Sapporo, Japan.

SOURCE: FREE RADICAL BIOLOGY AND MEDICINE, (1996) 21 (6) 755-61. Journal code: 8709159. ISSN: 0891-5849.

PUB. COUNTRY: United States

DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English

FILE SEGMENT: Priority Journals

ENTRY MONTH: 199702

ENTRY DATE: Entered STN: 19970305
Last Updated on STN: 19980206
Entered Medline: 19970218

ABSTRACT:
In the present study we demonstrated the protective effects of the **spin -trapping** agent alpha-phenyl-tert-butyl nitrone (PBN) against fulminant hepatitis with jaundice in LEC rats. In LEC rats an excess amount of copper is accumulated in the liver and causes hepatitis with severe jaundice. PBN was subcutaneously administered every 2 d at the concentration of 128 mg/kg, beginning with 13-week-old rats and continuing for 17 weeks. PBN prevented the loss of body weight, reduced death rate, and suppressed the increase in GTP and GOT values reflecting hepatic cell destruction. Ocular inspection also confirmed the suppressive effects of PBN on jaundice. In parallel with these phenomena, the amounts of thiobarbituric acid-reactive substances (TBARS) in livers of PBN-administered rats were found to be lower than those of non-PBN-administered rats. Little histological changes were observed in PBN-administered rats in comparison with non-PBN-administered rats. The protective effect of PBN on the formation of oxidative damage in liver DNA

was observed but not so remarkable as that on lipid peroxidation. From these results, it was concluded that PBN had the liver-protective effects against fulminant hepatitis with jaundice. This suggested that free radicals play an important role in abnormally accumulated copper-induced liver injury and that PBN potentially has therapeutic value for the treatment of hepatitis.

CONTROLLED TERM: Check Tags: Animal; Support, Non-U.S. Gov't
Aging
Alanine Transaminase: BL, blood
Aspartate Aminotransferases: BL, blood
*Copper
DNA: ME, metabolism
Deoxyguanosine: AA, analogs & derivatives
Deoxyguanosine: ME, metabolism
*Hepatitis, Toxic: PC, prevention & control
**Jaundice: CI, chemically induced**
***Jaundice: PC, prevention & control**
Lipid Peroxidation
Liver: ME, metabolism
*Nitrogen Oxides: TU, therapeutic use
Rats
Rats, Mutant Strains
*Spin Labels
Thiobarbituric Acid Reactive Substances: ME, metabolism
Weight Loss

CAS REGISTRY NO.: **3376-24-7 (phenyl-N-tert-butylnitrone)**; 7440-50-8 (Copper); 88847-89-6 (8-oxo-7-hydrodeoxyguanosine); 9007-49-2 (DNA); 961-07-9 (Deoxyguanosine)

CHEMICAL NAME: 0 (Nitrogen Oxides); 0 (Spin Labels); 0 (Thiobarbituric Acid Reactive Substances); EC 2.6.1.1 (Aspartate Aminotransferases); EC 2.6.1.2 (Alanine Transaminase)

L113 ANSWER 31 OF 44 MEDLINE

ACCESSION NUMBER: 96272734 MEDLINE

DOCUMENT NUMBER: 96272734 PubMed ID: 8696987

TITLE: Endotoxin-induced oxidative stress in the rat small intestine: role of nitric oxide.

AUTHOR: Chamulitrat W; Skrepnik N V; Spitzer J J

CORPORATE SOURCE: Department of Physiology, Louisiana State University Medical Center, New Orieans 70112-1393, USA.

CONTRACT NUMBER: AA09803 (NIAAA)

SOURCE: SHOCK, (1996 Mar) 5 (3) 217-22.
Journal code: 9421564. ISSN: 1073-2322.

PUB. COUNTRY: United States

DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English

FILE SEGMENT: Priority Journals

ENTRY MONTH: 199609

ENTRY DATE: Entered STN: 19960912
Last Updated on STN: 19960912
Entered Medline: 19960904

ABSTRACT:
Reactive oxygen species have been implicated in the gastrointestinal pathogenesis of septic and endotoxic shock. The objective of this study was to investigate the role of inducible nitric oxide synthase during endotoxin-induced formation of oxidants by cells of the small intestine. After intravenous Escherichia coli lipopolysaccharide (LPS) (1 mg/kg) injection, nitric oxide production was measured as nitrosyl complex formation in the ileum using electron paramagnetic resonance spectroscopy. Oxidative stress biomarkers were determined as duodenal mucosal-reduced thiols, the ileal lipid peroxidation and luminal free radical production using **spin** ***trapping*** methodology. Demonstration of nitrosyl complex formation commenced at 3 h and diminished 24 h post-LPS. Mucosal thiol levels were

decreased at 3, 6, 12, and 18 h post-LPS treatment. At these time point, the ileal lipid peroxidation also increased as did luminal formation of hydroxyl radical adduct. Nitric oxide synthase inhibitors reversed the elevation of hydroxyl radical formation and reversed the decrease in mucosal-reduced thiol levels in the LPS-treated rats. Our data indicate that nitric oxide or its oxidant product(s), such as peroxynitrite, contribute to oxidative injury in the small intestine of rats treated with endotoxin.

CONTROLLED TERM: Check Tags: Animal; Male; Support, U.S. Gov't, P.H.S.
Analysis of Variance
Cyclic N-Oxides
Enzyme Induction
Free Radicals
Intestine, Small: CY, cytology
*Intestine, Small: DE, drug effects
Intestine, Small: ME, metabolism
*Lipopolysaccharides: PD, pharmacology
Nitric Oxide: BI, biosynthesis
*Nitric Oxide: PH, physiology
*Nitric-Oxide Synthase: BI, biosynthesis
***Oxidative Stress: DE, drug effects**
Rats
Rats, Sprague-Dawley
Spin Labels

CAS REGISTRY NO.: 10102-43-9 (Nitric Oxide); **3317-61-1 (5,5-dimethyl-1-pyrroline-1-oxide)**

CHEMICAL NAME: 0 (Cyclic N-Oxides); 0 (Free Radicals); 0 (Lipopolysaccharides); 0 (Spin Labels); EC 1.14.13.39 (Nitric-Oxide Synthase)

L113 ANSWER 32 OF 44 MEDLINE
ACCESSION NUMBER: 93358345 MEDLINE
DOCUMENT NUMBER: 93358345 PubMed ID: 8394776
TITLE: Free radical formation in murine skin treated with tumour promoting organic peroxides.
AUTHOR: Timmins G S; Davies M J
CORPORATE SOURCE: Department of Chemistry, University of York, UK.
SOURCE: CARCINOGENESIS, (1993 Aug) 14 (8) 1499-503.
Journal code: 8008055. ISSN: 0143-3334.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199309
ENTRY DATE: Entered STN: 19931008
Last Updated on STN: 19931008
Entered Medline: 19930921

ABSTRACT:
The generation of free radicals from tumour-promoting organic peroxides applied to intact murine skin samples has been studied by EPR spectroscopy using two techniques: first direct observation of ascorbyl radicals produced from reactions of peroxide-related radicals with ascorbate, an important endogenous antioxidant, and secondly, observation of radical adducts produced by ***spin*** **-trapping**. Free radical generation from tumour-promoting organic peroxides can be seen to occur in intact skin tissue through a one-electron reductive pathway, and takes place at sites including the viable cells of the epidermis and/or dermis. This radical generation is dependent upon the penetration of the skin by the peroxides, with the stratum corneum representing a major diffusional barrier to their penetration of skin. The technique of using ascorbyl radical measurement by EPR spectroscopy as a means of studying and quantifying radical production in intact tissues, developed in this work, may prove of much use in the study of many free radicals and their reactions in a wide range of biological systems, particularly skin. When

combined with appropriate **spin-trapping** techniques, which enable identification of radical species and elucidation of their mechanisms of production, this enables the direct, real-time observation of radical reactions and mechanisms not previously possible in intact tissue samples.

CONTROLLED TERM: Check Tags: Animal; In Vitro; Male; Support, Non-U.S. Gov't
Ascorbic Acid: ME, metabolism
Cyclic N-Oxides
Dehydroascorbic Acid: AA, analogs & derivatives
Dehydroascorbic Acid: ME, metabolism
Electron Spin Resonance Spectroscopy
Free Radicals: ME, metabolism
Free Radicals: TO, toxicity
Mice
*Peroxides: ME, metabolism
*Peroxides: TO, toxicity
*Skin: DE, drug effects
***Skin Neoplasms: CI, chemically induced**
Spin Labels

CAS REGISTRY NO.: **3317-61-1 (5,5-dimethyl-1-pyrroline-1-oxide);** 490-83-5 (Dehydroascorbic Acid); 50-81-7 (Ascorbic Acid); 6730-29-6 (semidehydroascorbic acid)

CHEMICAL NAME: 0 (Cyclic N-Oxides); 0 (Free Radicals); 0 (Peroxides); 0 (Spin Labels)

L113 ANSWER 33 OF 44 EMBASE COPYRIGHT 2003 ELSEVIER SCI. B.V.

ACCESSION NUMBER: 2002407666 EMBASE

TITLE: Hydroperoxide formation in model collagens and collagen type I.

AUTHOR: Madison S.A.; McCallum J.E.B.; Rojas Wahl R.U.

CORPORATE SOURCE: R.U. Rojas Wahl, UCLA, Department of Chemistry, Box 951569, Los Angeles, CA 90095-1569, United States.
roy.rojas-wahl@unilever.com

SOURCE: International Journal of Cosmetic Science, (2002) 24/1 (43-52).
Refs: 46
ISSN: 0142-5463 CODEN: IJCMDW

COUNTRY: United Kingdom

DOCUMENT TYPE: Journal; Article

FILE SEGMENT: 013 Dermatology and Venereology
029 Clinical Biochemistry

LANGUAGE: English

SUMMARY LANGUAGE: English

ABSTRACT:

Protein hydroperoxides represent a relatively new concept in understanding biological oxidation chemistry. Here, we show with post-column-chemiluminescence that this sometimes remarkably stable and yet reactive species can be formed in collagen models and collagen type I when submitted to oxidative stress as exemplified by the Fenton reaction. These findings are supported by mass spectrometry and iodometry. Using (Proline-hydroxyproline-glycine)(10) (POG)(10), those hydroperoxides are stable for hours at room temperature and can give rise to free radicals in the presence of ferrous sulphate, as evidenced by EPR **spin trapping** with DMPO. Possible implications for biological systems are discussed with emphasis on collagen in the extracellular matrix in skin as a major type of connective tissue.

CONTROLLED TERM: Medical Descriptors:
model
chemoluminescence
**oxidative stress**
Fenton reaction
mass spectrometry

room temperature
electron spin resonance
extracellular matrix
**skin**
hydrolysis
derivatization
liquid chromatography
article
Drug Descriptors:
*hydroperoxide
*collagen type 1
proline
hydroxyproline
glycine
free radical
ferrous sulfate
5,5 dimethyl 1 pyrroline 1 oxide

CAS REGISTRY NO.: (proline) 147-85-3, 7005-20-1; (hydroxyproline) 51-35-4, 6912-67-0; (glycine) 56-40-6, 6000-43-7, 6000-44-8; (ferrous sulfate) 10028-21-4, 10124-49-9, 13463-43-9, 7720-78-7, 7782-63-0; (5,5 dimethyl 1 pyrroline 1 oxide) **3317-61-1**

L113 ANSWER 34 OF 44 EMBASE COPYRIGHT 2003 ELSEVIER SCI. B.V.

ACCESSION NUMBER: 2001340434 EMBASE

TITLE: Time dependent amelioration against ischemic brain damage by glial cell line-derived neurotrophic factor after transient middle cerebral artery occlusion in rat.

AUTHOR: Zhang W.R.; Hayashi T.; Iwai M.; Nagano I.; Sato K.; Manabe Y.; Abe K.

CORPORATE SOURCE: K. Abe, Department of Neurology, Okayama University Medical School, 2-5-1 Shikatacho, Okayama 700-8558, Japan. zhang@cc.okayama-u.ac.jp

SOURCE: Brain Research, (8 Jun 2001) 903/1-2 (253-256).
Refs: 19
ISSN: 0006-8993 CODEN: BRREAP

PUBLISHER IDENT.: S 0006-8993(01)02364-2

COUNTRY: Netherlands

DOCUMENT TYPE: Journal; Article

FILE SEGMENT: 008 Neurology and Neurosurgery
030 Pharmacology
037 Drug Literature Index

LANGUAGE: English

SUMMARY LANGUAGE: English

ABSTRACT:
Time dependent influence of glial cell line-derived neurotrophic factor (GDNF) was examined after 90 min of transient middle cerebral artery occlusion (MCAO) in rats. Treatment with GDNF significantly reduced the infarct volume stained with 2,3,5-triphenyltetrazolium chloride (TTC) when GDNF was topically applied at 0 and 1 h of reperfusion, but became insignificant at 3 h as compared to vehicle group. The protective effect of GDNF was closely related to the significant reduction of the number of terminal deoxynucleotidyl transferase-mediated dUTP-biotin in situ nick end labeling (TUNEL) positive cells as well as immunofluorescently positive cells for active forms of caspases, especially active caspase-3 but not -9. Thus, the present study showed that topical application of GDNF significantly reduced infarct size in a time-dependent manner, while the therapeutic time window was shorter than other chemical compounds such as an NMDA receptor antagonist (MK-801) and a free radical scavenger (alpha-phenyl-tert-butyl-nitrone, PBN). The effect of GDNF was stronger in suppressing active caspase-3 than active caspase-9. .COPYRGT. 2001 Elsevier Science B.V. All rights reserved.

CONTROLLED TERM: Medical Descriptors:

*brain ischemia: DT, drug therapy
*middle cerebral artery occlusion: DT, drug therapy
chronotherapy
drug effect
staining
dose time effect relation
nick end labeling
immunofluorescence
enzyme activity
enzyme inhibition
drug activity
reperfusion
nonhuman
male
rat
animal experiment
animal model
controlled study
animal tissue
article
priority journal
Drug Descriptors:
*glial cell line derived neurotrophic factor: CM, drug comparison
*glial cell line derived neurotrophic factor: DV, drug development
*glial cell line derived neurotrophic factor: DO, drug dose
*glial cell line derived neurotrophic factor: DT, drug therapy
*glial cell line derived neurotrophic factor: PD, pharmacology
***glial cell line derived neurotrophic factor: TP, topical drug administration**
triphenyltetrazolium
DNA nucleotidylexotransferase
deoxyuridine triphosphate derivative
biotin
caspase: EC, endogenous compound
caspase 3: EC, endogenous compound
caspase 9: EC, endogenous compound
chemical compound: CM, drug comparison
n methyl dextro aspartic acid receptor blocking agent: CM, drug comparison
dizocilpine: CM, drug comparison
scavenger: CM, drug comparison
n tert butyl alpha phenylnitrone: CM, drug comparison

CAS REGISTRY NO.: (triphenyltetrazolium) 298-96-4; (DNA nucleotidylexotransferase) 9027-67-2; (biotin) 58-85-5; (caspase) 186322-81-6; (caspase 3) 169592-56-7; (caspase 9) 180189-96-2; (dizocilpine) 77086-21-6; (n tert butyl alpha phenylnitrone) **3376-24-7**

CHEMICAL NAME: Mk 801

L113 ANSWER 35 OF 44 EMBASE COPYRIGHT 2003 ELSEVIER SCI. B.V.
ACCESSION NUMBER: 1999166731 EMBASE
TITLE: Thalidomide on the comeback trail.
AUTHOR: Hales B.F.
CORPORATE SOURCE: B.F. Hales, Dept. of Pharmacol. and Therapeutics, McGill University, 3655 Drummond Street, Montreal, Que. H3G 1Y6, Canada. bhales@pharma.mcgill.ca
SOURCE: Nature Medicine, (1999) 5/5 (489-490).
Refs: 11
ISSN: 1078-8956 CODEN: NAMEFI

COUNTRY: United States
DOCUMENT TYPE: Journal; (Short Survey)
FILE SEGMENT: 037 Drug Literature Index
052 Toxicology
LANGUAGE: English
SUMMARY LANGUAGE: English
ABSTRACT:
Will new insights into Thalldomide's teratogenic mechanism help make its return a safe one?.

CONTROLLED TERM: Medical Descriptors:
*teratogenicity: ET, etiology
***erythema nodosum leprosum: DT, drug therapy**
drug sensitivity
species difference
**oxidative stress**
DNA damage
human
nonhuman
short survey
priority journal
Drug Descriptors:
*thalidomide: DT, drug therapy
*thalidomide: TO, drug toxicity
n tert butyl alpha phenylnitrone
buthionine sulfoximine
DNA
transcription factor
reactive oxygen metabolite
CAS REGISTRY NO.: (thalidomide) 50-35-1; (n tert butyl alpha phenylnitrone) **3376-24-7**; (buthionine sulfoximine) 5072-26-4; (DNA) 9007-49-2

L113 ANSWER 36 OF 44 EMBASE COPYRIGHT 2003 ELSEVIER SCI. B.V.
ACCESSION NUMBER: 1999281087 EMBASE
TITLE: Alpha lipoic acid (ALA) protects proteins against the hydroxyl free radical-induced alterations: Rationale for its geriatric topical application.
AUTHOR: Perricone N.; Nagy K.; Horvath F.; Dajko G.; Uray I.; Zs.-Nagy I.
CORPORATE SOURCE: I. Zs.-Nagy, Department of Gerontology, University Medical School, POB 50, H-4012 Debrecen, Hungary
SOURCE: Archives of Gerontology and Geriatrics, (1999) 29/1 (45-56).
Refs: 39
ISSN: 0167-4943 CODEN: AGGEDL
PUBLISHER IDENT.: S 0167-4943(99)00022-9
COUNTRY: Ireland
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 020 Gerontology and Geriatrics
029 Clinical Biochemistry
037 Drug Literature Index
LANGUAGE: English
SUMMARY LANGUAGE: English
ABSTRACT:
The well known OH. free radical scavenging properties of .alpha.-lipoic acid (ALA) cannot be easily utilized for biological experiments, because the compound is practically insoluble in water. We elaborated a simple method of preparing its Na-salt (Na-ALA) which proved to be water soluble. It has been demonstrated by ESR **spin trapping** experiments with DMPO, using the Fenton reaction as the source of OH. free radicals that Na-ALA maintains its OH. free radical scavenging ability: it reacts nearly an order of magnitude faster with these radicals than the **spin trap**

itself. It was tested in two different systems to determine whether Na-ALA was able to protect bovine serum albumin (BSA) against the OH. free radical-induced polymerization and protein oxidation. (i) OH. free radicals were generated by Fenton reaction in the presence of BSA. This protein is polymerized by these radicals shown by the loss of its water solubility; Na-ALA exerted a considerable protective effect against this type of protein damage. (ii) BSA oxidation was induced by Co-gamma irradiation of 80 krad, resulting in a strong increase in the protein carbonyl content. Na-ALA inhibited this carbonyl formation very efficiently. The data suggest that the interaction of the OH radical with Na-ALA takes place on the disulfide group, yielding thiosulfinate or thiosulfonate. The results indicate that the geriatric topical application of Na-ALA may have an established rationale. Copyright (C) 1999 Elsevier Science Ireland Ltd.

CONTROLLED TERM: Medical Descriptors:
*drug solubility
*drug synthesis
*structure activity relation
*protein polymerization
**topical drug administration**
**spin trapping**
nonhuman
controlled study
article
priority journal
Drug Descriptors:
*free radical: EC, endogenous compound
*antioxidant: DV, drug development
*thioctic acid: DV, drug development
hydrogen peroxide: EC, endogenous compound
bovine serum albumin
carbonyl derivative: EC, endogenous compound

CAS REGISTRY NO.: (thioctic acid) 1077-29-8, 1200-22-2, 2319-84-8, 62-46-4; (hydrogen peroxide) 7722-84-1

L113 ANSWER 37 OF 44 EMBASE COPYRIGHT 2003 ELSEVIER SCI. B.V.
ACCESSION NUMBER: 1999004331 EMBASE
TITLE: Protection against aminoglycoside otic drop-induced ototoxicity by a **spin trap**: 1. Acute effects.
AUTHOR: Hester T.O.; Jones R.O.; Clerici W.J.
CORPORATE SOURCE: Dr. W.J. Clerici, Department of Surgery, Div. of Otolaryngol.-Head/Neck Surg., Chandler Medical Center, Lexington, KY 40536-0084, United States
SOURCE: Otolaryngology - Head and Neck Surgery, (1998) 119/6 (581-587).
Refs: 48
ISSN: 0194-5998 CODEN: OTOLDL
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 011 Otorhinolaryngology
037 Drug Literature Index
LANGUAGE: English
SUMMARY LANGUAGE: English
ABSTRACT:
Topical administration of aminoglycoside antibiotic drops containing neomycin and polymyxin B disrupts cochlear structure and function in rodents, possibly as a result of reactive oxygen species generation. This study investigated the ability of a **spin trap**, .alpha.-phenyl-tert-butyl-nitrone (PBN), to prevent acute aminoglycoside antibiotic drop-induced cochlear dysfunction. Guinea pigs were monitored for compound action potential thresholds and 1.0 .mu.V root-mean-square cochlear microphonic isopotential curve values, then injected intraperitoneally with PBN (60 mg/kg) or saline

solution. After 10 minutes, 50 .mu.l of PBN (100 mmol/L) or artificial perilymph was applied to the round window membrane, followed after 10 minutes with artificial perilymph or aminoglycoside antibiotic drops (50 .mu.l). From 10 to 60 minutes after exposure, mean compound action potential thresholds progressively increased in the artificial perilymph-aminoglycoside antibiotic drop group, beginning with high frequencies and later including ever-lower frequencies. These threshold shifts in compound action potentials were significantly greater ($p < 0.05$) than those seen in the artificial perilymph-artificial perilymph or PBN-aminoglycoside antibiotic drop groups. This finding indicates that PBN provided protection against acute aminoglycoside antibiotic drop-induced compound action potential threshold sensitivity loss. Mean cochlear microphonic shift values at 60 minutes in the artificial perilymph-aminoglycoside antibiotic drop group significantly exceeded those of the other groups only at the highest frequencies. These data suggest that acute aminoglycoside antibiotic drop-induced cochlear disruption primarily affects high frequency compound action potential function and may be partially reactive oxygen species-mediated and preventable.

CONTROLLED TERM: Medical Descriptors:
*ototoxicity
*cochlea injury
oxidative stress
guinea pig
cochlea fenestra
dose response
perilymph
nonhuman
animal experiment
animal model
controlled study
intraperitoneal drug administration
**topical drug administration**
article
Drug Descriptors:
*ear drops: AD, drug administration
*aminoglycoside antibiotic agent: AD, drug administration
*aminoglycoside antibiotic agent: DV, drug development
*aminoglycoside antibiotic agent: TO, drug toxicity
*n tert butyl alpha phenylnitrone: AD, drug administration
*n tert butyl alpha phenylnitrone: DV, drug development
*n tert butyl alpha phenylnitrone: DO, drug dose
*n tert butyl alpha phenylnitrone: PD, pharmacology
neomycin: AD, drug administration
neomycin: DV, drug development
neomycin: TO, drug toxicity
polymyxin b: AD, drug administration
polymyxin b: DV, drug development
polymyxin b: TO, drug toxicity
hydrocortisone

CAS REGISTRY NO.: (n tert butyl alpha phenylnitrone) **3376-24-7**; (neomycin) 11004-65-2, 1404-04-2, 1405-10-3, 8026-22-0; (polymyxin b) 1404-26-8, 1405-20-5; (hydrocortisone) 50-23-7

COMPANY NAME: Schein

L113 ANSWER 38 OF 44 EMBASE COPYRIGHT 2003 ELSEVIER SCI. B.V.

ACCESSION NUMBER: 1998158508 EMBASE

TITLE: Release of nitric oxide from a spin trap, N-tert-butyl-.alpha.- phenylnitrone, under various oxidative conditions.

AUTHOR: Saito K.; Yoshioka H.; Kazama S.; Cutler R.G.

CORPORATE SOURCE: K. Saito, Gerontology Research Center, National Institute on Aging, NIH, 4940 Eastern Avenue, Baltimore, MD 21224,

United States
SOURCE: Biological and Pharmaceutical Bulletin, (1998) 21/4 (401-404).
Refs: 21
ISSN: 0918-6158 CODEN: BPBLEO
COUNTRY: Japan
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 030 Pharmacology
037 Drug Literature Index
LANGUAGE: English
SUMMARY LANGUAGE: English
ABSTRACT:
Nitric oxide (NO) generation from a spin trap, N-tert-butyl-.alpha.-phenylnitrone (PBN) under various oxidative conditions was examined. The absorbance of PBN at 295 nm decreased with time of UV-irradiation, showing that PBN was decomposed by UV irradiation. The hydroxyl radical formed from a Fenton reagent also decomposed PBN, but there was little effect by a peroxyl radical and a superoxide. Nitrite, an oxidative product of NO, in PBN solution was determined using a NOx analyzer based on Griess reaction. UV- irradiation and the hydroxyl radical also formed nitrite. Direct detection of NO from the sample on reaction with hydroxyl radical was successful using a GC/MS/SIM on the UV-irradiated sample. NO generated in PBN solutions activated guanylate cyclase. From these results, PBN is viewed as a new kind of medicine which acts as an antioxidant and as an NO donor in vivo.

CONTROLLED TERM: Medical Descriptors:
*oxidation
***oxidative stress**
**ultraviolet radiation**
enzyme activity
gas chromatography
mass spectrometry
electron spin resonance
decomposition
article
Drug Descriptors:
*n tert butyl alpha phenylnitrone: PD, pharmacology
*nitric oxide
guanylate cyclase
cyclic gmp
CAS REGISTRY NO.: (n tert butyl alpha phenylnitrone) **3376-24-7;** (nitric oxide) 10102-43-9; (guanylate cyclase) 9054-75-5; (cyclic gmp) 7665-99-8

L113 ANSWER 39 OF 44 EMBASE COPYRIGHT 2003 ELSEVIER SCI. B.V.
ACCESSION NUMBER: 95124454 EMBASE
DOCUMENT NUMBER: 1995124454
TITLE: In vivo detection of anthralin-derived free radicals in the skim of hairless mice by low-frequency electron paramagnetic resonance spectroscopy.
AUTHOR: Mader K.; Bacic G.; Swartz H.M.
CORPORATE SOURCE: Department of Radiology, Dartmouth Medical School, Strasenburgh 308,Hanover, NH 03755, United States
SOURCE: Journal of Investigative Dermatology, (1995) 104/4 (514-517).
ISSN: 0022-202X CODEN: JIDEAE
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 013 Dermatology and Venereology
030 Pharmacology
037 Drug Literature Index
LANGUAGE: English
SUMMARY LANGUAGE: English

ABSTRACT:
Free radicals were directly detected in vivo in the skin of hairless mice by low-frequency electron paramagnetic resonance spectroscopy after topical application of anthralin under pertinent therapeutic conditions. The electron paramagnetic resonance signal intensity increased steadily, reaching a maximum after about 1 d and decreased slowly in the following days, probably because of desquamation of the skin. We conclude from the spectroscopic features (single line with a line width of 6 gauss; g = 2.0036) and from the pharmacokinetic pattern that the observed signal arises from the final products of anthralin metabolism (ether-insoluble polymeric structures-'anthralin brown'). Two potential antioxidants, vitamin E and the **spin trap** tert-butylphenylnitrone, decreased the amount of the anthralin-derived radical that was formed. Neither vitamin E radicals nor tert-butylphenylnitrone spin adducts were observed. We suggest that electron paramagnetic resonance is a valuable tool for the noninvasive and direct in vitro monitoring of drug-induced radical formation in the skin under therapeutic conditions.

CONTROLLED TERM: Medical Descriptors:
***oxidative stress**
***skin defect: DI, diagnosis**
animal experiment
animal model
animal tissue
article
controlled study
electron spin resonance
male
mouse
nonhuman
priority journal
**topical drug administration**
Drug Descriptors:
*alpha tocopherol: PD, pharmacology
*alpha tocopherol: IT, drug interaction
*alpha tocopherol: CB, drug combination
*alpha tocopherol: CM, drug comparison
*dithranol: TO, drug toxicity
*dithranol: IT, drug interaction
*dithranol: CB, drug combination
*dithranol: PK, pharmacokinetics
*free radical
*n tert butyl alpha phenylnitrone: PD, pharmacology
*n tert butyl alpha phenylnitrone: IT, drug interaction
*n tert butyl alpha phenylnitrone: CB, drug combination
*n tert butyl alpha phenylnitrone: CM, drug comparison

CAS REGISTRY NO.: (alpha tocopherol) 1406-18-4, 1406-70-8, 52225-20-4, 58-95-7, 59-02-9; (dithranol) 1143-38-0, 480-22-8; (n tert butyl alpha phenylnitrone) **3376-24-7**

L113 ANSWER 40 OF 44 EMBASE COPYRIGHT 2003 ELSEVIER SCI. B.V.
ACCESSION NUMBER: 94239107 EMBASE
DOCUMENT NUMBER: 1994239107
TITLE: Structure-activity relationships for the formation of secondary radicals and inhibition of keratinocyte proliferation by 9-anthrones.
AUTHOR: Hayden P.J.; Free K.E.; Chignell C.F.
CORPORATE SOURCE: Laboratory of Molecular Biophysics, NIEHS, Mail Drop 17-05, P.O. Box 12233,Research Triangle Park, NC 27709, United States
SOURCE: Molecular Pharmacology, (1994) 46/1 (186-198).
ISSN: 0026-895X CODEN: MOPMA3
COUNTRY: United States
DOCUMENT TYPE: Journal; Article

FILE SEGMENT: 013 Dermatology and Venereology
030 Pharmacology
037 Drug Literature Index
LANGUAGE: English
SUMMARY LANGUAGE: English
ABSTRACT:

The biological properties of tumor-promoting and antipsoriatic 9- anthrones have been hypothesized to be mediated by free radical products such as the corresponding 9-anthron-10-yl radicals or by O2/+, OH, and other persistent secondary radicals that are formed in the skin after topical treatment with 9-anthrones. To gain additional insights into the possible role of reactive oxygen or secondary radicals in mediating the biological effects of 9-anthrones, we have used EPR spectroscopy to investigate the formation of these species by a series of 9-anthrones or 9-anthrone dimers with known tumor-promoting and antipsoriatic activities. The effect of the 9- anthrones on keratinocyte proliferation in vitro was also investigated. 5,5-Dimethyl-1-pyrroline N-oxide was used as a **spin trap** to detect reactive oxygen-centered radicals in aqueous buffer/dimethylsulfoxide solutions. Superoxide was trapped during the autoxidation of most of the 9-anthrones. For 9-anthrones that generated no detectable superoxide, evidence of anthronyl-peroxyl radical formation was found instead. In the presence of Fe3+ complexed to EDTA, but not diethylenetriaminepentaacetic acid, the hydroxyl radical was produced by all of the 9-anthrones. 9-Anthrone dimers produced oxygen-centered radicals only weakly or not at all. Direct EPR was used to detect 9-anthrone-derived secondary radicals in keratinocyte suspensions or in dimethylsulfoxide solutions. These radicals were similar to those previously reported to occur in skin after topical treatment with the antipsoriatic drug anthralin (1,8-dihydroxy-9-anthrone). In contrast to the ubiquitous ability of the 9-anthrones to generate reactive oxygen radicals, only the hydroxy-substituted 9-anthrones or their dimers possessed significant secondary radical-forming ability. The ability of the 9-anthrones or dimers to form secondary radicals in keratinocytes was found to correlate with their in vitro inhibition of keratinocyte proliferation. The data suggest the possible importance of reactive dimeric intermediates in mediating the biological effects of the 9-anthrones.

CONTROLLED TERM: Medical Descriptors:
*cell proliferation
*keratinocyte
*psoriasis: DT, drug therapy
*skin carcinogenesis
animal cell
article
controlled study
drug mechanism
drug structure
electron spin resonance
inflammation
mouse
nonhuman
skin irritation
structure activity relation
**topical drug administration**
Drug Descriptors:
*9 anthroic acid: AD, drug administration
*9 anthroic acid: AN, drug analysis
*9 anthroic acid: DT, drug therapy
*9 anthroic acid: PD, pharmacology
*dithranol: AD, drug administration
*dithranol: AN, drug analysis
*dithranol: DT, drug therapy
*dithranol: PD, pharmacology
*oxygen radical

*reactive oxygen metabolite
5,5 dimethyl 1 pyrroline 1 oxide
CAS REGISTRY NO.: (9 anthroic acid) 723-62-6; (dithranol) 1143-38-0, 480-22-8; (5,5 dimethyl 1 pyrroline 1 oxide) **3317-61-1**

L113 ANSWER 41 OF 44 EMBASE COPYRIGHT 2003 ELSEVIER SCI. B.V.
ACCESSION NUMBER: 93218834 EMBASE
DOCUMENT NUMBER: 1993218834
TITLE: MPP+ and MPDP+ induced oxygen radical formation with mitochondrial enzymes.
AUTHOR: Adams Jr. J.D.; Klaidman L.K.; Leung A.C.
CORPORATE SOURCE: Dept.Molecular Pharmacol./Toxicology, School of Pharmacy, University of Southern California,Los Angeles, CA 90033, United States
SOURCE: Free Radical Biology and Medicine, (1993) 15/2 (181-186). ISSN: 0891-5849 CODEN: FRBMEH
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 029 Clinical Biochemistry
LANGUAGE: English
SUMMARY LANGUAGE: English
ABSTRACT:
MPP+ has been reported to inhibit reduced nicotinamide adenine dinucleotide (NADH) dehydrogenase in mitochondria, which results in the formation of O2.-. The current report demonstrates that H2O2 and HO. are also products of MPP+ interaction with NADH dehydrogenase. It is possible that MPP. formation precedes the formation of some of these active oxygen species. Reducing equivalents for radical formation come from NADH. MPP+ may be capable of interacting with submitochondrial particles at a site other than the rotenone site, which results in some formation of oxygen radicals. Plasma amine oxidase incubations with MPDP+ resulted in O2.-, H2O2, and perhaps HO. formation. This is probably due to MPP. formation from the oxidation of MPDP+. This study presents new findings that indicate the potential importance of oxygen radical formation in mitochondria during MPTP toxicity.

CONTROLLED TERM: Medical Descriptors:
*heart mitochondrion
***oxidative stress**
*parkinson disease: ET, etiology
animal cell
article
controlled study
cow
nonhuman
priority journal
**ultraviolet radiation**
Drug Descriptors:
*1 methyl 4 phenylpyridinium: TO, drug toxicity
*5,5 dimethyl 1 pyrroline 1 oxide
*hydrogen peroxide: EC, endogenous compound
*manganese sulfate
*oxidoreductase
*pentetic acid
*rotenone
*superoxide dismutase
*xanthine
CAS REGISTRY NO.: (1 methyl 4 phenylpyridinium) 39794-99-5, 48134-75-4; (5,5 dimethyl 1 pyrroline 1 oxide) **3317-61-1**; (hydrogen peroxide) 7722-84-1; (manganese sulfate) 10124-55-7, 7785-87-7; (oxidoreductase) 9035-73-8, 9035-82-9, 9037-80-3, 9055-15-6; (pentetic acid) 14047-41-7, 67-43-6; (rotenone) 83-79-4; (superoxide

dismutase) 37294-21-6, 9016-01-7, 9054-89-1; (xanthine) 69-89-6

L113 ANSWER 42 OF 44 EMBASE COPYRIGHT 2003 ELSEVIER SCI. B.V.
ACCESSION NUMBER: 93218833 EMBASE
DOCUMENT NUMBER: 1993218833
TITLE: Redox cycling of MPP+: Evidence for a new mechanism involving hydride transfer with xanthine oxidase, aldehyde dehydrogenase, and lipoamide dehydrogenase.
AUTHOR: Klaidman L.K.; Adams Jr. J.D.; Leung A.C.; Kim S.S.; Cadenas E.
CORPORATE SOURCE: Dept.Molecular Pharmacol./Toxicology, School of Pharmacy, University of Southern California,Los Angeles, CA 90033, United States
SOURCE: Free Radical Biology and Medicine, (1993) 15/2 (169-179). ISSN: 0891-5849 CODEN: FRBMEH
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 029 Clinical Biochemistry
LANGUAGE: English
SUMMARY LANGUAGE: English
ABSTRACT:
MPP+ is redox active in the presence of cytochrome P450 reductase and induces the formation of O2.- and HO.. In this study, we report the redox cycling capability of MPP+ with additional enzymes and with UV photolysis detected through ESR techniques. The treatment of MPP+ with UV light resulted in the production of HO. trapped as a spin adduct. Two of the enzymes examined in this study, xanthine oxidase and aldehyde dehydrogenase, produced O2.- in the presence of substrate. However, when MPP+ was added to the incubations, the radical trapped by DMPO was HO.. This indicates that MPP+ redox cycles in the presence of these two enzymes or UV light, which produces HO.. Our data also suggest that MPP+ is reduced by lipoamide dehydrogenase. MPP+ stimulated the oxidation of reduced nicotinamide adenine dinucleotide (NADH) by the enzyme at concentrations between 2 mM and 8 mM of MPP+. Higher concentrations of MPP+ inhibited lipoamide dehydrogenase. MPP+ appears to be redox active with a number of redox enzymes. The mechanism involved may be hydride transfer from the enzymes to MPP+, rather than a direct single-electron reduction.

CONTROLLED TERM: Medical Descriptors:
***oxidative stress**
***ultraviolet radiation**
article
controlled study
priority journal
Drug Descriptors:
*1 methyl 4 phenylpyridinium: TO, drug toxicity
*5,5 dimethyl 1 pyrroline 1 oxide
*aldehyde dehydrogenase
*oxygen radical: EC, endogenous compound
*reduced nicotinamide adenine dinucleotide: EC, endogenous compound
*superoxide: EC, endogenous compound
*superoxide dismutase
*xanthine oxidase
1 methyl 4 phenylpyridine derivative: TO, drug toxicity
unclassified drug
CAS REGISTRY NO.: (1 methyl 4 phenylpyridinium) 39794-99-5, 48134-75-4; (5,5 dimethyl 1 pyrroline 1 oxide) **3317-61-1**; (aldehyde dehydrogenase) 37353-37-0, 9028-86-8; (reduced nicotinamide adenine dinucleotide) 58-68-4; (superoxide) 11062-77-4; (superoxide dismutase) 37294-21-6, 9016-01-7, 9054-89-1; (xanthine oxidase) 9002-17-9

L113 ANSWER 43 OF 44 EMBASE COPYRIGHT 2003 ELSEVIER SCI. B.V.
ACCESSION NUMBER: 92203735 EMBASE
DOCUMENT NUMBER: 1992203735
TITLE: Essential oil phenyl propanoids. Useful as .cntdot.OH scavengers?.
AUTHOR: Taira J.; Ikemoto T.; Yoneya T.; Hagi A.; Murakami A.; Makino K.
CORPORATE SOURCE: Cosmetics Laboratory, Kanebo Ltd., 5-3-28 Kotobuki-cho,Odawara, Kanagawa 250, Japan
SOURCE: Free Radical Research Communications, (1992) 16/3 (197-204).
ISSN: 8755-0199 CODEN: FRRCEX
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 030 Pharmacology
037 Drug Literature Index
LANGUAGE: English
SUMMARY LANGUAGE: English

CONTROLLED TERM: Medical Descriptors:
*antioxidant activity
animal tissue
article
chemical reaction kinetics
electron spin resonance
male
nonhuman
rat
**skin defect: PC, prevention**
**ultraviolet radiation**
Drug Descriptors:
*essential oil
*hydroxyl radical
*phenol derivative
*scavenger
5,5 dimethyl 1 pyrroline 1 oxide
isoeugenol
thiobarbituric acid
CAS REGISTRY NO.: (hydroxyl radical) 3352-57-6; (5,5 dimethyl 1 pyrroline 1 oxide) **3317-61-1**; (isoeugenol) 97-54-1; (thiobarbituric acid) 504-17-6
COMPANY NAME: Takasago (Japan)

L113 ANSWER 44 OF 44 EMBASE COPYRIGHT 2003 ELSEVIER SCI. B.V.
ACCESSION NUMBER: 88166543 EMBASE
DOCUMENT NUMBER: 1988166543
TITLE: Ability of N-tert-butyl alpha phenylnitrone (PBN) to be used in isolated perfused heart **spin trapping** experiments: Preliminary studies.
AUTHOR: Charlon V.; De Leiris J.
CORPORATE SOURCE: Laboratoire de Physiopathologie du Metabolisme Cardiaque, Universite Scientifique Technologique, Grenoble, France
SOURCE: Basic Research in Cardiology, (1988) 83/3 (306-313).
ISSN: 0300-8428 CODEN: BRCAB7
COUNTRY: Germany
DOCUMENT TYPE: Journal
FILE SEGMENT: 018 Cardiovascular Diseases and Cardiovascular Surgery
030 Pharmacology
037 Drug Literature Index
LANGUAGE: English
SUMMARY LANGUAGE: English
ABSTRACT:
The aim of this study was to investigate the possibility of oxygen-free radical

***spin*** **trapping** with PBN, in models of isolated perfused hearts. Preliminary studies reported here demonstrate that (i) PBN may be precisely measured with UV spectroscopy, (ii) commercially available PBN does not show any ESR signal, (iii) PBN does not trap significant amounts of free radicals in a perfusion medium oxygenated for at least 3 h, and (iv) when added at 15 or 56 mM in the perfusion medium, PBN is a highly toxic compound, whereas no toxic effect was observed with 3mM-containing perfusate.

CONTROLLED TERM: Medical Descriptors:
*coronary reperfusion
*heart muscle ischemia
biological model
spectroscopy
**topical drug administration**
Drug Descriptors:
*free radical
*n tert butyl alpha phenylnitrone
CAS REGISTRY NO.: (n tert butyl alpha phenylnitrone) **3376-24-7**
COMPANY NAME: Aldrich

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DICTIONARY FILE UPDATES: 7 APR 2003 HIGHEST RN 502131-66-0

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http://www.cas.org/ONLINE/STN/STNOTES/stnotes27.pdf

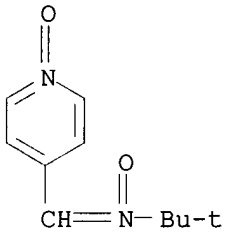
=> s 3376-24-7 or 3317-61-1 or 2564-83-2 or 66893-81-0

1 3376-24-7
(3376-24-7/RN)
1 3317-61-1
(3317-61-1/RN)
1 2564-83-2
(2564-83-2/RN)
1 66893-81-0
(66893-81-0/RN)
L114 4 3376-24-7 OR 3317-61-1 OR 2564-83-2 OR 66893-81-0

=> d ide 1-4; fil hom

L114 ANSWER 1 OF 4 REGISTRY COPYRIGHT 2003 ACS
RN **66893-81-0** REGISTRY
CN 2-Propanamine, 2-methyl-N-[(1-oxido-4-pyridinyl)methylene]-, N-oxide (9CI) (CA INDEX NAME)

OTHER CA INDEX NAMES:
CN 2-Propanamine, 2-methyl-N-(4-pyridinylmethylene)-, N,N'-dioxide
OTHER NAMES:
CN .alpha.-(4-Pyridyl-1-oxide)-N-tert-butylnitrone
CN 4-POBN
CN C-(4-Pyridinyl-N-oxide)-N-tert-butylnitrone
CN N-tert-Butyl-.alpha.-(4-pyridyl-1-oxide) nitrone
CN POBN
FS 3D CONCORD
DR 83016-64-2
MF C10 H14 N2 O2
CI COM
LC STN Files: AGRICOLA, BEILSTEIN*, BIOSIS, CA, CAPLUS, CASREACT, CHEMCATS, CHEMLIST, CSCHEM, MSDS-OHS, NIOSHTIC, TOXCENTER, USPATFULL
(*File contains numerically searchable property data)
Other Sources: EINECS**
(**Enter CHEMLIST File for up-to-date regulatory information)

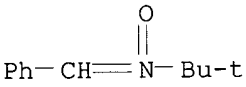



**PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT**

144 REFERENCES IN FILE CA (1962 TO DATE)
12 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
143 REFERENCES IN FILE CAPLUS (1962 TO DATE)

L114 ANSWER 2 OF 4 REGISTRY COPYRIGHT 2003 ACS
RN **3376-24-7** REGISTRY
CN 2-Propanamine, 2-methyl-N-(phenylmethylene)-, N-oxide (9CI) (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN Nitrone, N-tert-butyl-.alpha.-phenyl- (6CI, 7CI, 8CI)
OTHER NAMES:
CN .alpha.-Phenyl-N-tert-butylnitrone
CN .alpha.-Phenyl-tertbutyl nitrone
CN 2-Methyl-N-(phenylmethylene)-2-propanamine N-oxide
CN 2-Phenyl-N-tert-butylnitrone
CN Benzylidene-tert-butylamine N-oxide
CN Benzylidene-tert-butylamine oxide
CN C-Phenyl-N-tert-butylnitrone
CN C-Phenyl-N-tert-butylnitrone
CN N-Benzylidene-tert-butylamine N-oxide
CN N-Benzylidene-tert-butylamine oxide
CN N-tert-Butyl-.alpha.-phenylnitrone
CN N-tert-Butyl-2-phenylnitrone
CN N-tert-Butyl-C-phenylnitrone
CN PBN
CN PBN (amine oxide)
CN tert-Butyl(benzylidene)amine N-oxide
FS 3D CONCORD
DR 165047-88-1, 173777-90-7, 50643-08-8, 68315-30-0, 154345-12-7, 115995-20-5

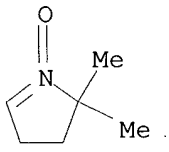
MF C11 H15 N O
CI COM
LC STN Files: AGRICOLA, BEILSTEIN*, BIOBUSINESS, BIOSIS, BIOTECHNO, CA, CANCERLIT, CAOLD, CAPLUS, CASREACT, CHEMCATS, CHEMINFORMRX, CHEMLIST, CSCHEM, CSNB, DDFU, DETHERM*, DRUGU, EMBASE, IPA, MEDLINE, MRCK*, MSDS-OHS, NIOSHTIC, RTECS*, SPECINFO, TOXCENTER, USPAT2, USPATFULL
(*File contains numerically searchable property data)
Other Sources: EINECS**, NDSL**, TSCA**
(**Enter CHEMLIST File for up-to-date regulatory information)



**PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT**

1007 REFERENCES IN FILE CA (1962 TO DATE)
24 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
1007 REFERENCES IN FILE CAPLUS (1962 TO DATE)
8 REFERENCES IN FILE CAOLD (PRIOR TO 1967)

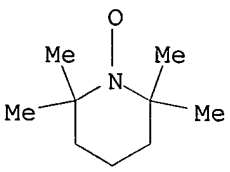
L114 ANSWER 3 OF 4 REGISTRY COPYRIGHT 2003 ACS
RN **3317-61-1** REGISTRY
CN 2H-Pyrrole, 3,4-dihydro-2,2-dimethyl-, 1-oxide (9CI) (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN 1-Pyrroline, 5,5-dimethyl-, 1-oxide (6CI, 7CI, 8CI)
OTHER NAMES:
CN 2,2-Dimethyl-3,4-dihydro-2H-pyrrole N-oxide
CN 5,5-Dimethyl-.DELTA.1-pyrroline 1-oxide
CN 5,5-Dimethyl-.DELTA.1-pyrroline N-oxide
CN 5,5-Dimethyl-1-pyrroline 1-oxide
CN 5,5-Dimethyl-1-pyrroline N-oxide
CN 5,5-Dimethyl-4,5-dihydro-3H-pyrrole N-oxide
CN DMPO
FS 3D CONCORD
MF C6 H11 N O
CI COM
LC STN Files: AGRICOLA, ANABSTR, BEILSTEIN*, BIOBUSINESS, BIOSIS, BIOTECHNO, CA, CANCERLIT, CAOLD, CAPLUS, CASREACT, CHEMCATS, CHEMINFORMRX, CHEMLIST, CSCHEM, EMBASE, IFICDB, IFIPAT, IFIUDB, MEDLINE, MRCK*, MSDS-OHS, PIRA, TOXCENTER, USPAT2, USPATFULL
(*File contains numerically searchable property data)
Other Sources: EINECS**
(**Enter CHEMLIST File for up-to-date regulatory information)



**PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT**

820 REFERENCES IN FILE CA (1962 TO DATE)
45 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
821 REFERENCES IN FILE CAPLUS (1962 TO DATE)
7 REFERENCES IN FILE CAOLD (PRIOR TO 1967)

L114 ANSWER 4 OF 4 REGISTRY COPYRIGHT 2003 ACS
RN (**2564-83-2**) REGISTRY
CN 1-Piperidinyloxy, 2,2,6,6-tetramethyl- (9CI) (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN Piperidinooxy, 2,2,6,6-tetramethyl- (7CI, 8CI)
OTHER NAMES:
CN 1,1,5,5-Tetramethylpentamethylene nitroxide
CN 1-Oxyl-2,2,6,6-tetramethylpiperidine
CN 2,2,6,6-Tetramethyl-1-oxylpiperidine
CN 2,2,6,6-Tetramethyl-1-piperadoxyl
CN 2,2,6,6-Tetramethyl-1-piperidinoxyl
CN 2,2,6,6-Tetramethyl-1-piperidinyloxy
CN 2,2,6,6-Tetramethyl-1-piperidyloxy
CN 2,2,6,6-Tetramethylpiperidin-1-oxy
CN 2,2,6,6-Tetramethylpiperidin-1-oxyl radical
CN 2,2,6,6-Tetramethylpiperidin-N-oxyl
CN 2,2,6,6-Tetramethylpiperidine N-oxide
CN 2,2,6,6-Tetramethylpiperidine N-oxide radical
CN 2,2,6,6-Tetramethylpiperidine N-oxy
CN 2,2,6,6-Tetramethylpiperidine N-oxyl
CN 2,2,6,6-Tetramethylpiperidine N-oxyl radical
CN 2,2,6,6-Tetramethylpiperidine nitroxide
CN 2,2,6,6-Tetramethylpiperidine nitroxide radical
CN 2,2,6,6-Tetramethylpiperidine oxide
CN 2,2,6,6-Tetramethylpiperidine-1-oxyl
CN 2,2,6,6-Tetramethylpiperidino-1-oxy
CN 2,2,6,6-Tetramethylpiperidinooxy
CN 2,2,6,6-Tetramethylpiperidinooxy radical
CN 2,2,6,6-Tetramethylpiperidinooxyl
CN 2,2,6,6-Tetramethylpiperidinoxyl
CN 2,2,6,6-Tetramethylpiperidinoxyl radical
CN 2,2,6,6-Tetramethylpiperidinyl 1-oxide
CN 2,2,6,6-Tetramethylpiperidinyl-1-oxyl
CN 2,2,6,6-Tetramethylpiperidinyl-N-oxy
CN 2,2,6,6-Tetramethylpiperidinyloxy
CN 2,2,6,6-Tetramethylpiperidoxyl
CN HO 6
CN Tanan
CN Tanane
CN Tempo
CN TEMPO
CN TMPO
DR 126517-51-9, 54637-06-8, 125012-91-1, 64104-42-3, 25657-03-8, 26933-82-4
MF C9 H18 N O
CI COM
LC STN Files: AGRICOLA, ANABSTR, BEILSTEIN*, BIOBUSINESS, BIOSIS, CA, CANCERLIT, CAOLD, CAPLUS, CASREACT, CEN, CHEMCATS, CHEMINFORMRX, CHEMLIST, CIN, CSCHEM, DDFU, DRUGU, GMELIN*, IFICDB, IFIPAT, IFIUDB, IPA, MEDLINE, MRCK*, NIOSHTIC, PIRA, PROMT, RTECS*, TOXCENTER, USPAT2, USPATFULL
(*File contains numerically searchable property data)
Other Sources: EINECS**, TSCA**
(**Enter CHEMLIST File for up-to-date regulatory information)



2357 REFERENCES IN FILE CA (1962 TO DATE)
100 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
2360 REFERENCES IN FILE CAPLUS (1962 TO DATE)
23 REFERENCES IN FILE CAOLD (PRIOR TO 1967)

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